



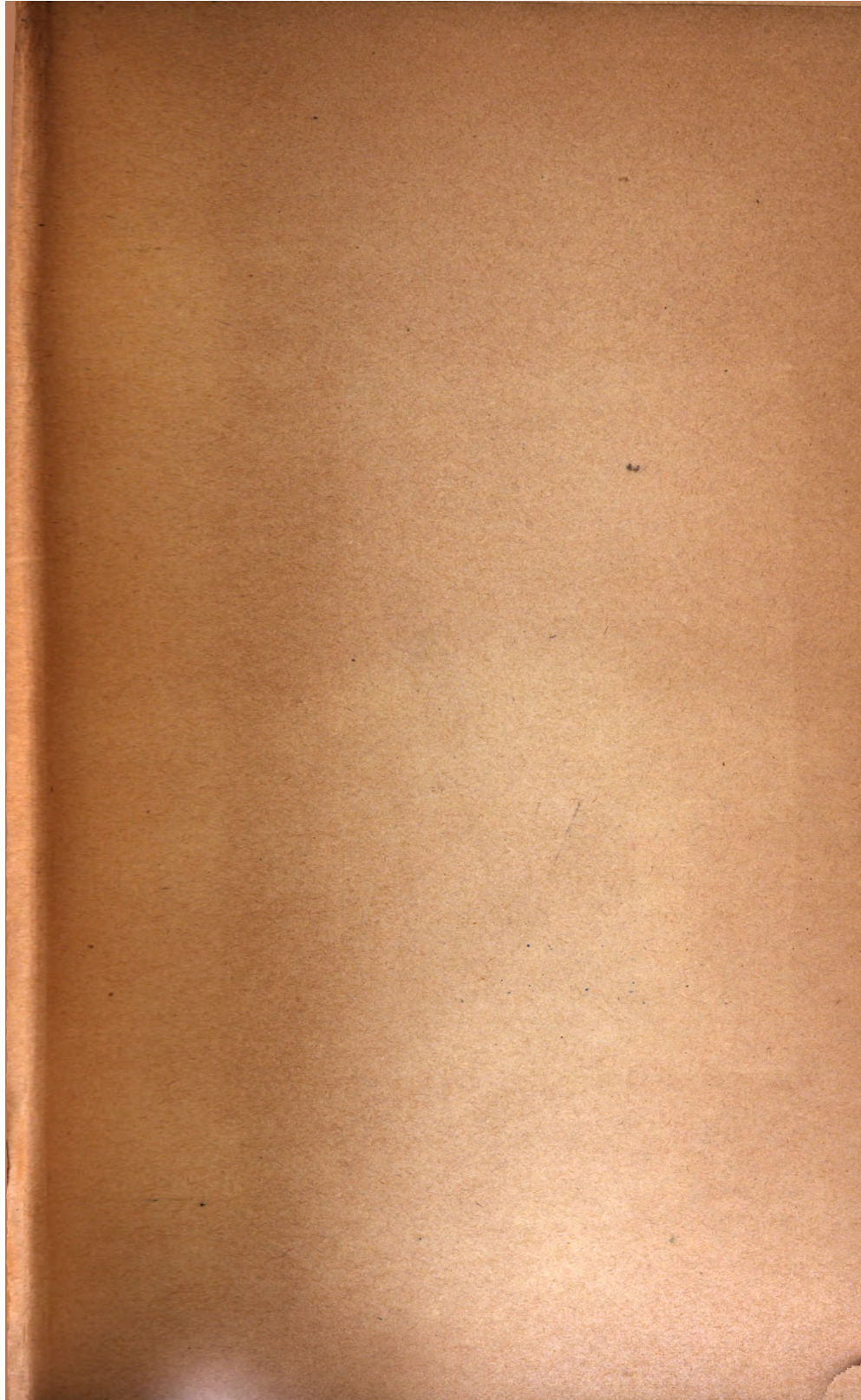
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GUY'S HOSPITAL REPORTS.

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AND

F. J. STEWARD, M.S.

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The Editors, The Medical Chronicle, The Owens College, Manchester

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COARCTATION OF THE AORTA

AS ILLUSTRATED BY CASES FROM THE POST-MORTEM RECORDS OF GUY'S HOSPITAL FROM 1826—1902.

By JOHN FAWCETT, M.D.

IN the work connected with re-cataloguing the museum I have recently met with several specimens of the condition which gives the title to this paper, and as the lesion is a rare and interesting one I have collected together the cases from our records. Some of them, to which reference will be made, have already been published in the transactions of various societies. The rarity of the lesion is evidenced by the fact that there are only eighteen cases of coarctation of aorta out of some 22,316 autopsies performed since 1826. The abstracts of the cases form an appendix to the paper.

Site and form of stricture.—The site of the lesion is variously described in the reports, but coincides with that noted by Peacock (!) and others, viz., at or near to the point where the ductus arteriosus joins the descending aorta. The subjoined table gives an analysis of the shape and degree of stenosis as detailed in the reports :—

	SITE.	SIZE AND FORM OF CONTRACTION.
CASE 1	Immediately above the ductus arteriosus.	"Very marked."
CASE 2	(Exact position not stated.)	Lumen admitted a fine probe only. Cone-like.
CASE 3	Immediately below the ductus arteriosus.	Complete occlusion. Cone-like.
CASE 4	Just beyond left subclavian.	Lumen admitted a fine probe only.
CASE 5	Lowest branchial arch beyond left subclavian.	Lumen measures $\frac{3}{8}$ inch in diameter.
CASE 6	1 inch below left subclavian.	Lumen measures $\frac{1}{2}$ inch in diameter or less.
CASE 7	At or just before the ductus arteriosus.	Admitted No. 8-10 catheter. Sudden annular stricture.
CASE 8	Just beyond the ductus arteriosus.	Admitted No. 6-8 catheter. "Conical in shape from left carotid artery onwards, and then became closely constricted."
CASE 9	At site of ductus arteriosus.	Lumen admitted an ordinary probe. Gradually narrowed into a conical shape.
CASE 10	Opposite or slightly above left subclavian. Just above ductus arteriosus.	"Tight narrow band more than $\frac{1}{2}$ the circumference of the vessel."
CASE 11	(Exact site not stated.)	"Narrowing much and contracting to a probe fissure."
CASE 12	Opposite abnormal point of origin of right subclavian, which was situated below the left subclavian.	A ridge-like narrowing.
CASE 13	To the left side of the left subclavian.	A well-marked contraction.
CASE 14	"Below the pit of the duct." ? Arteriosus.	A partial thickish ridge.
CASE 15	Immediately to the right of the ductus arteriosus.	Lumen less than $\frac{1}{2}$ inch in diameter.
CASE 16	Near the former opening of the ductus arteriosus.	(No note of.)
CASE 17	1 inch beyond the left subclavian.	Sudden annular constriction about one line in breadth.
CASE 18	Beyond ductus arteriosus.	Decidedly small aorta.

It will be seen in the table that in only one case (No. 3) was the aorta completely obliterated, while in the others the lumen varied from half an inch in diameter to one which would admit the passage of a fine probe only. The narrowing is seen to take the form either of an abrupt annular constriction, or of a gradual diminution in size in the form of a cone.

The proportion of examples of occlusion is, in this series, much smaller than in Peacock's original collection of cases, among which ten out of forty showed complete obliteration of the canal. Lee Dickinson and Fenton (*) state that out of one hundred and five cases there were fourteen examples of complete occlusion of the vessel.

Condition of ductus arteriosus.—It is stated by most writers on this subject that the duct is closed in the larger number of cases, but the present series does not support that statement. In nine out of the eighteen cases this point is not referred to, but in six of the remaining nine the duct was found to be open.

Condition of the heart and its valves, and of the aorta :—

(i.) *Heart.*—In twelve cases the heart was enlarged, with a general increase in size in seven, viz. (Nos. 2, 9, 10, 11, 12, 16 and 17). In two cases (Nos. 7 and 15) the left ventricle was chiefly affected, and in one case (No. 8) the right ventricle. In two others (Nos. 13 and 14) the right side was apparently alone involved; in No. 13, there were several malformations present and no doubt the right side of the heart had had most of the work to do during life, the left side being small, the aorta narrow and its walls small and thin. In No. 14, the details of the autopsy are too meagre to allow any conclusion to be drawn as to the cause of the enlargement of the right side.

On reference to the cases it may be seen that in several of them the increase in size was considerable, *e.g.* :—

Case 2	weight, 23 ounces.
" 8	" 17 "
" 10	" 18 "

In three cases (Nos. 3, 5 and 6) there was no marked enlargement of the heart, although in Nos. 3 and 6, it was probably somewhat larger than normal. In the remaining three

cases the associated congenital malformations and consequent early death, are such as to exclude them from consideration.

From the above analysis it is clear that the block in the aorta has led to a dilatation and hypertrophy of the heart such as might result from obstruction at the aortic orifice. Peacock found the same result, the heart being uniformly and considerably enlarged in eighteen out of forty cases, while in ten others there was a moderate increase in size affecting chiefly the left ventricle. In only two cases of his series is the heart stated to have been quite healthy. These results have been confirmed by later observers, thus showing that, in a large proportion of the cases, coarctation of the aorta does give rise to enlargement of the heart.

(ii.) *Valves.*—The constant strain to which the aortic valves are subjected leads in the majority of cases to a chronic inflammatory change, with the result that the valves become thickened, or adherent, or the orifice stenosed. For example, in no less than ten cases (Nos. 3, 7, 8, 9, 10, 11, 12, 14, 15 and 17) were the aortic valves affected, and in six of them the mitral valve showed a similar change. In only three cases (Nos. 2, 5 and 6) were the valves apparently healthy. In one case (No. 16) there is no note of the condition of the valves, and in another (No. 18) the aortic valves were thin and ill developed, the main portion of the work of the heart having been performed by the right ventricle.

The cases (Nos. 1, 4 and 18) are omitted from consideration in this connection on account of the many congenital malformations which existed.

(iii.) *Aorta.*—The aorta on the proximal side of the stricture has been described as dilated in most cases, *e.g.*, in Peacock's series, out of the twenty-nine cases in which the condition of the vessel is mentioned, it was found to be of natural size and free from disease in three only. In the present series the aorta was little affected, for in only four of the eighteen cases (Nos. 8, 12, 14 and 16) is there any record of a dilatation of that vessel. The degree of dilatation, too, can only have been slight, for the terms used in describing it in the reports are

"full large," "little dilated," "dilated to end of arch," and "wide in ascending part," respectively.

Again, in many of the previously recorded cases the vessel on the proximal side is described as being thickened and atheromatous, but in the "Guy's" series this does not appear to have been so, in that it was only noted in five cases (Nos. 2, 3, 5, 6 and 10). In cases Nos. 7 and 8, Dr. Goodhart suggested that the reason for this part of the aorta being so little damaged was that the tension in the vessel was less on account of the chronic endocarditis present. In this connection it is interesting to note that in Cases Nos. 2, 5 and 6 the valves were healthy, while in Nos. 8 and 10 aortic stenosis was present.

On the distal side of the stricture the aorta has been noticed to either rapidly dilate so as to become larger than usual, and then to gradually lessen in size again, or to remain small throughout. The coats also are sometimes thinner than normal and aneurysmal dilatations may be present. In the present series there is no mention of a dilatation of the aorta immediately below the stricture, but in three cases an aneurysm was present.

In Case No. 5, death was due to the rupture of a small aneurysm on the proximal side of the obstruction. In Case No. 7, the aorta is described as having cracked over a patch of atheroma below the constriction, and given rise to a dissecting aneurysm in an early stage of development. In Case No. 14, a larger saccular aneurysm arose from the ascending portion of the aorta.

In Cases Nos. 3, 7, 8, 9, 10, 12 and 13, the descending aorta was small, and in Nos. 10 and 13, the description is such as to indicate that this portion of the vessel was narrow throughout. In only three cases was the wall of the vessel thin, viz., in Nos. 3 and 8 below the constriction, and in No. 17 above it. There is no note as to the size of the aorta in Cases Nos. 2, 5, 6, 14, 15 and 16.

As the lumen of the main trunk of the aorta is more or less obstructed in the different cases, so in order to maintain an adequate circulation through the body the blood must pass through other channels which, as a result, become enlarged.

The anastomotic connections have been shown to be chiefly through some of the branches of the subclavian artery with branches arising from the aorta below the site of constriction. These vessels are the transverse cervical and the posterior scapular branches of the thyroid axis; the subscapular, the superior intercostal, the aortic intercostals, the internal mammary and its branches, in particular the superior epigastric in its anastomoses with the deep epigastric branch of the external iliac artery.

In this collection of cases the collateral circulation has been only imperfectly dealt with, but in Cases Nos. 2, 3, 7, 8, 9, 11, 13 and 17, the following vessels were found enlarged and in some cases tortuous, viz., the large vessels from the arch, the scapular, the superior intercostal, the internal mammary and the iliac arteries. The anastomoses are well shown in a specimen taken from the dissecting room and preserved in the museum; it was fully described by Dr. Hale White (*). The subject was a man *æt.* 46 years, thought during life to have heart disease only.

In all the cases of the "Guy's" series in which the collateral circulation was well developed it may be seen (*v.* Table p. 2) that the constriction was considerable and was associated either with enlargement of the heart, or changes in the aortic valves, or both.

Associated malformations.—In eight out of the eighteen cases various congenital malformations were present. They were most numerous in Cases Nos. 1, 4 and 18, the coarctation of the aorta having in consequence no special bearing on the clinical history of the cases.

In Case No. 1, the heart was much enlarged, especially the right ventricle, the ductus arteriosus was patent, the septum of the ventricles imperfect, and the mitral orifice stenosed. The stomach, intestines, pancreas and spleen were transposed and the right kidney was situated much lower down than normal.

In Case No. 4, the foramen ovale and ductus arteriosus were patent, the ventricular septum was imperfect, the two carotids and the left subclavian arteries arose directly from the aorta,

but the right subclavian artery could not be found. There was no direct communication between the right auricle and ventricle.

In Case No. 18, the aorta and pulmonary artery were transposed and in addition to other peculiarities, the septum between the auricles, as well as that of the ventricles, was perforated.

In Case No. 9, the mitral valves were malformed, the muscle columns being absent, and at the intermembranous septum a pouch projected into the right ventricle.

In Case No. 10, a miniature additional valve was present in the aorta just above the valves.

In Case No. 12, four vessels arose from the aorta, viz., the right and left carotid, the left and right subclavian arteries, in the order named from right to left. The right subclavian artery arose from the posterior part of the descending portion of the arch, crossed the vertebral column behind the œsophagus, and so passed onwards to its usual destination.

In Case No. 13, the foramen ovale was patent, and posterior to it the septum was deficient. The right pulmonary veins opened into the right auricle. The aorta was narrow and its valves thin.

In Case No. 15, the anterior portion of the falx cerebri was wanting. The left kidney was only half the usual size, while the right was twice as large as normal. A large branch from the middle of the basilar artery ran into the cavernous sinus.

The large proportion of associated congenital malformations is referred to by many previous writers as a frequent accompaniment of coarctation of the aorta, and the present series of cases fully confirms the statement.

Age and sex.—Peacock states that the cases are met with most frequently during the active period of adult life. This is borne out by the "Guy's" series, for out of the eighteen cases, twelve, or 66 per cent., were between the ages of twenty-one and fifty years at the time of death. The youngest of the whole number was sixteen hours and the oldest sixty-four years. In Peacock's series the relation of males to females was nearly four to one, but in the present series they are almost equally

divided, viz., nine males to eight females, the sex in one case not being stated.

Clinical features and diagnosis.—This collection of cases may be divided up clinically into three groups :—

1. Those with physical signs indicative of some cardiac lesion.
2. Those without any symptoms of a cardiac lesion, or of a lesion of the circulatory system.
3. Those who died early and in whom there were many congenital malformations present.

Group 1, contains the cases of most interest from the clinical side, and includes Cases Nos. 2, 3, 7, 8, 9, 10 and 16.

(In Cases Nos. 12, 13, 14 and 15, no clinical history was available; but in No. 12, in association with the enlargement of the heart, it was noticed at the autopsy that there was œdema of the legs and scrotum.)

In not one of the cases, however, were any physical signs detected during life affording a clue to the actual condition which existed, *e.g.*—

Case No. 2; was thought to be suffering from a chronic interstitial nephritis.

Case No. 3; the diagnosis was that of mitral stenosis and regurgitation, in addition to disease of the aortic valves.

Case No. 7; the symptoms suggested a renal origin rather than a cardiac one.

Cases Nos. 8, 9 and 16, were apparently in the last stage of chronic heart disease.

Case No. 10; the bruits heard were not typical of any particular lesion, but Mr. Wilkinson King thought that they indicated the presence of a patent ductus arteriosus.

In Group 2 (including Cases Nos. 5, 6, 11, and 17), death ensued from various causes.

Case No. 5; sudden death from rupture of a small aneurysm on the proximal side of the obstruction.

Case No. 6; death resulted from an epithelioma of the tongue, with dysphagia and bronchitis.

Case No. 11; death occurred from cerebral hæmorrhage.

Case No. 17; death resulted from acute pericarditis and jaundice associated with an impacted gall-stone.

In Group 3 (including Cases Nos. 1, 4 and 18), death was the result, either directly or indirectly, of the various malformations which existed.

From the above analysis it is seen that some of the cases remained latent throughout, while in others the patients were thought to be the subjects of a chronic heart lesion. In a few recorded instances a correct diagnosis has been arrived at by the discovery of enlarged and tortuous vessels on the trunk, in particular of the epigastric, the posterior scapular, and internal mammary arteries. Thus, although the disease is so rare, yet a diagnosis would be quite possible in the presence of a well developed collateral circulation.

Other signs to which attention has been drawn by previous observers are:

(i.) Purring sensation felt in the varicose vessels; a bruit heard over them.

(ii.) The pulse in the abdominal aorta or in the lower extremities, may be feeble, slow, or delayed.

(iii.) Special cardiac murmurs, *e.g.*, a systolic bruit on the left side behind, in the region of the fourth dorsal vertebra, and transmitted downwards between it and the left scapula.

(iv.) Dilatation of the arch of the aorta, which may be felt in the suprasternal notch.

Ætiology.—In that the stenosis or obliteration of the aorta in these cases is always situated at or about the same place, and that the coats of the aorta are not necessarily diseased, previous writers have, for the most part, regarded the lesion as due to a developmental defect. Peacock is of this opinion and points to the large proportion of associated congenital malformations which he says, "would scarcely be met with in an equal number of cases of any form of accidental disease." This latter point is well exemplified in the present series (*vide Associated Malformations*).

The actual method of production is somewhat doubtful, but the most commonly accepted explanation is, that it is due to the defective development of the portions of the branchial arches which go to form the aorta, between the left subclavian artery and the ductus arteriosus, this part of the vessel not expanding and developing in the usual way.

In the cases in which the ductus arteriosus remains patent the narrowing of the aorta must have been sufficient at birth to produce some obstruction to the blood-flow into the descending aorta, and so some of it reached that vessel through the ductus arteriosus. In those cases in which the ductus arteriosus is closed, the obstruction of the aorta at birth cannot have been very great, but what probably happens in these cases is that the affected portion of the vessel does not expand with the growth of the body generally, and so an obstruction is gradually produced. This latter process, as Peacock observes, is very favourable to the development of a well-marked collateral circulation, and when this is complete the aortic constriction will be no longer kept expanded by the blood passing through it, but will contract and may ultimately result in a complete occlusion of the aorta at the narrowed part.

The other theories that have been put forward are :—

- (i.) That the obstruction of the vessel is due to organisation of a thrombus formed during the closure of the ductus arteriosus, the process spreading on to the aorta.
- (ii.) That the stenosis is the result of an extension of the process, by means of which the ductus arteriosus is closed, into the aorta.

Peacock points out that in relation to theory (i.), the process of obliteration of the duct does not depend upon closure by a thrombus, but by a gradual thickening and contraction of its coats, commencing at the aortic extremity. With regard to theory (ii.) he also says that the explanation suggested is insufficient to account for those cases in which the ductus arteriosus remains patent, and also that it would not coincide with the path of closure of the ducts which commences at its aortic and not at its pulmonary extremity.

In the Cases Nos. 7 and 8, Dr. Goodhart⁽⁴⁾ suggests that the coarctation is the result of a chronic inflammatory change connected with the closure of the ductus arteriosus. The ductus arteriosus was very thick in both of these cases, and in the second case there was a considerable amount of fibrous tissue around it. In most of the cases recorded there is, however, little or no evidence of inflammatory change, and it would appear possible that the thickening noted in these two cases may have been the result of a somewhat excessive proliferation of the tissues of the vessel wall during the process of closure of the duct, or during the progressive narrowing of the aorta, following the establishment of the collateral circulation.

REFERENCES.

1. Peacock, *British and Foreign Medico-Chirurgical Review*, vol. xxv., 1860, p. 467.
2. Lee Dickinson and Fenton, *Lancet*, October 27th, 1900.
3. Hale White, W., *Trans. Patholog. Soc.*, vol. xxxvi., 1885.
4. Goodhart, J. F., *Trans. Patholog. Soc.*, vol. xxvi., 1875.
5. Wilks, S., *Trans. Patholog. Soc.*, vol. x., 1859.
6. Babington, W., *Trans. Patholog. Soc.*, vol. i., 1847.
7. Quincke, *Diseases of Arteries*, *Ziemssen's Cyclopædia*, vol. vi., p. 473.

APPENDIX OF CASES.

In each case the reference in brackets at the head of the report applies to the post-mortem records of Guy's Hospital.

In abstracting the cases, the words used by the reporter in describing the cases have, as far as possible, been adhered to, even though many of the terms are not now in use.

CASE 1 (*see Insp.*, 1901, No. 375).—Grace S., æt. 5 months, was admitted under Dr. Hale White for cough and difficulty in breathing of six weeks' duration. The child had previously suffered from measles, bronchitis, diarrhoea and vomiting. On admission, respiration was very rapid, and there were signs of a general bronchitis. A systolic bruit was heard in the pulmonary area. There was no cyanosis nor anasarca. Autopsy: The heart was enlarged. The ventricles were hypertrophied, especially the right. Dr. Bryant states that the heart was quite double the size of that of a seven months' child he had examined the same afternoon. A very marked coarctation of the aorta was present, immediately above the point of junction of the ductus arteriosus. The ductus arteriosus was patent. There was a communication between the two ventricles, both at the upper and the lower part of the septum. The mitral valve was thickened and stenosed, and the chordæ tendineæ also thickened. The left auricle was hypertrophied, and the right auricle very large. The aortic and pulmonary valves were healthy. The stomach, intestines, pancreas and spleen were transposed, the spleen being situated beneath the right lobe of the liver. The right kidney was much lower than normal, being on a level with the prominence of the sacrum. There was a diffuse broncho-pneumonia with collapse distributed over the posterior third of both lower lobes.

CASE 2 (*see Insp.*, 1899, No. 142).—Thomas M., æt. 45, was admitted under Dr. Pye-Smith for dyspnoea and oedema of the lower extremities. He was said to have had "rheumatic gout" seven years before, which confined him to bed for two months. He, however, recovered from this except for a slight cough and palpitation. In October, 1897, he is stated to have been treated by a doctor for an enlarged heart, and in October, 1898, he was admitted into Guy's Hospital with effusion into the right pleura. The arteries were calcareous, the left ventricle was hypertrophied. After returning to work in January, 1899, the "heart" symptoms became more marked, and were associated with palpitation, and oedema of the legs. There was a history of venereal disease. On admission, he was orthopnoic, the pulse-rate could not be counted, and there was an apical systolic bruit. The patient died quite suddenly while sitting in front of the fire. Autopsy: The heart weighed 23 ounces. The auricles were dilated and the ventricles both dilated and hypertrophied. The

aortic valves were competent, but their edges, perhaps, were a little thickened. All the other valves were healthy. The pulmonary artery was large and its wall hypertrophied. Aorta: The ascending portion of the arch was atheromatous, large, hard, calcareous plates being present. There was a very marked narrowing of the aorta, so as only to just admit a fine probe. (There is no note as to the exact position of the constriction nor as to whether the ductus arteriosus was patent or not.) Enlargement, atheroma and tortuosity of the scapular arteries was present, the vessels being as big as a normal radial. The spleen was small—2½ ounces. The kidneys contained some infarcts. The liver, which was “nutmegged,” showed some slight scarring and puckering on its upper surface. The pleuræ were thickened and adhesions were present. The lungs were œdematous and part of the right lung was carnified. The branches of the pulmonary artery were thickened, dilated and atheromatous.

CASE 3 (*see Insp.*, 1899, No. 441).—Eliza S., æt. circa 45, was admitted under Dr. Horrocks for pregnancy associated with heart disease. She had born eight live children, the last confinement having taken place a year previously. On admission, the patient was very dyspnoic, and had bronchitis and albuminuria. The physical signs were thought to indicate disease of both the aortic and mitral valves. The day following admission labour was induced as the patient's condition was critical. She did not improve after this, but during the next fortnight the œdema of the legs increased, and she lapsed into a semi-comatose condition, with twitchings of the hands and face and incontinence of urine and feces. Autopsy: The heart was very little, if any, larger than normal. The aortic valves were thickened and irregular, and the orifice stenosed, two of the valves having coalesced. The neighbouring portion of the aorta was atheromatous. Attached to the ventricular aspect of one of the cusps was an organised pyramidal-shaped mass of fibrin 2·25 centimetres in length. All the other valves were normal. The aorta was completely occluded immediately below the point of junction of the ductus arteriosus with the aorta. Above the occlusion the left subclavian and common carotid were seen to be dilated, and as large as the innominate artery, the subclavian artery being the largest of them all. From the origin of the left subclavian the aorta gradually contracted, being conical in shape, down to the occluded part. Below this the thoracic aorta was diminished in size by about one half, the abdominal aorta was still smaller, and the coats, although thin, were healthy in appearance. The ductus arteriosus was patent. The lungs were indurated, the liver was “nutmegged,” the spleen firm, and the kidneys tough and congested.

CASE 4 (*see Insp.*, 1893, No. 100).—A female child, æt. 16 hours, admitted under Mr. Davies-Colley, with a ventral hernia, which contained a large part of the small intestine. The sac was opened, the intestine returned and the opening closed with sutures. The child became cyanosed and convulsed, and died the same evening. Autopsy: The heart was broader than normal, the right side being thicker than the left. The right ventricle was small, the left auricle very large, at least six times the volume of the right. The foramen ovale was patent. There was no direct communication between the right auricle and ventricle. The septum ventriculorum was imperfect, and the left ventricle was small and contracted. The pulmonary and mitral valves

were rather thickened, and minute vegetations were present upon them. The aorta arose as usual by a large trunk from which sprung the two carotid arteries and the left subclavian artery, but Dr. Pitt, who made the autopsy, was unable to find the right subclavian artery. Just beyond the origin of the left subclavian artery the vessel was contracted so as only to admit a fine probe, and beyond this again the aorta was dilated.

CASE 5 (*see Insp.*, 1886, No. 75).—Emily M., æt. 41, was admitted under Dr. Pavy for pain in the back between the scapulæ. The heart and lung sounds were normal. Shortly after admission the house-physician was called to see the patient and found her in a collapsed and pulseless condition. She died two hours later. She was a book-felder, and the report states that there was some reason to suspect a toper. Autopsy: One and a quarter pints of blood were found in the left pleura; the whole mediastinum was infiltrated with blood together with the connective tissue at the root of the neck and base of the heart. One ounce, or more, of liquid blood was present in the pericardium. Dr. Goodhart, who made the post-mortem examination, states that "the aorta was rather atheromatous, especially about the distal half of the transverse arch, and the situation of the lowest branchial arch, beyond the left subclavian was obviously more than usually contracted. It did not approach any extreme degree of coarctation, being three-eighths of an inch in diameter, and it could not have caused any obstruction for the heart only weighed six ounces. Nor was the aorta at all dilated." The valves were healthy. About half an inch on the proximal side of the contraction, below and to the left of the left subclavian artery there was a round opening, which Dr. Goodhart thought was most probably a small aneurysm of the aorta, and that this had ruptured and formed a dissecting aneurysm tearing across the thoracic intercostals and cesophageal branches in its course. From this opening as far as the coeliac axis the blood had dissected downwards, apparently in the external coat, but in some places the wall between the proper channel and the new one was so thin that it was almost through again into the normal channel. The abdominal aorta was extensively diseased, as were also the coronary arteries of the heart and the iliac arteries. The coeliac axis and other abdominal vessels were all thick and soft, also the arteries of the neck.

CASE 6 (*see Insp.*, 1885, No. 201).—Geo. Y., æt. 64, was admitted under Dr. Pye-Smith for an epithelioma of the tongue involving the larynx. He had had dysphagia for three months. On admission, he was dyspnoic, and respiration was of a wheezy character. Death ensued shortly afterwards, being preceded by delirium at night. There is no record of any cardiac lesion, nor of the patient having suffered from any symptoms indicative of heart disease. Autopsy: The heart weighed 11 ounces and was normal. In the descending portion of the aorta, an inch from the left subclavian artery, the vessel was stenosed, measuring at this point half an inch in diameter or rather less. The three trunks from the arch were atheromatous, and in the abdominal aorta there was a similar degeneration, with calcareous deposits. The cerebral arteries were thickened and the main branches of the abdominal aorta showed atheromatous degeneration. The lungs were very emphysematous, the tubes dilated, and the branches of the pulmonary artery thickened. The pleura was thickened, and there were secondary deposits of growth on this membrane as well as in the bronchial and cervical glands and in the liver.

CASE 7 (*see Insp.*, 1875, No. 37, and *Trans. Pathological Soc.*, vol. xxvi., 1875).—James M., æt. 27, was admitted under Dr. Wilks for dropsy and deficient respiratory murmur at the base of the right lung. His occupation had been that of a carpenter. Nine weeks previously his face and eyelids had become swollen, and his urine scanty and black. The cardiac impulse was situated an inch outside and below the nipple. A loud apical systolic bruit was audible, traceable towards the axilla and up into the aortic area, and described as post-diastolic in rhythm. After admission it disappeared from the front of the chest. There was one-third of albumen present in the urine. Death was gradual, being due to exhaustion associated with a restless delirium. Autopsy: There was moderate œdema of the legs and much ascites. The right pleura was full of fluid, and the left contained some fluid, as well as recent lymph on the surface. The heart weighed fourteen and a half ounces, the left ventricle being extremely hypertrophied. Its cavity contained a small amount of ante-mortem coagulum. The mitral valve was thick and rather small, with recent vegetations on the auricular surface. The two anterior cusps of the aortic valve were adherent, and from the angle of adhesion sprang a tough fibrinous mass the size of a pea. The posterior cusp was somewhat thickened. The aorta above the constriction was rather thick and of full calibre, though not remarkably dilated. At the point of junction of the ductus arteriosus the vessel was suddenly constricted as if a cord had been tied round it, the lumen of this portion being the size of a No. 8 or 10 gum elastic catheter. Below the constriction the vessel had cracked over an atheromatous patch about two-thirds of an inch in length. The breach of surface was situated at the closed end of the ductus arteriosus, and an early dissecting aneurysm had formed between it and the left branch of the pulmonary artery. The branches of the thyroid axis were all large, especially the superior intercostal. The internal mammary was also large, and the anastomoses between the epigastric arteries were well marked and the vessels tortuous. The ductus arteriosus was closed. There was a chronic tubal nephritis present, and infarctions of the spleen.

CASE 8 (*see Insp.*, 1875, No. 122, and *Trans. Pathological Soc.*, vol. xxvi., 1875).—William McH., æt. 37, was admitted under Dr. Wilks. About six or seven years before he had had an attack of bronchitis lasting six or seven weeks. Three years ago he had had gonorrhœa and a swollen testicle. At Christmas, 1874, he became ill, with increasing weakness, and from the first some swelling of the abdomen. There was a month's history of swelling of the legs and face. Ten days before admission he developed a right hemiplegia. On admission, there was slight swelling of both ankles and the legs and chest were œdematous; the face was yellowish and the capillaries on the cheeks congested. There was some paresis of the tongue on the right side and a sensation of numbness. The chest was narrow and there was œdema of the bases of the lungs. The area of præcordial dulness was large; the cardiac impulse was situated two inches below and half an inch external to the nipple. Heart's action irregular and quick, and sounds muffled. An apical systolic bruit was heard and a doubtful pericardial rub. The liver and spleen were enlarged. The urine contained about three-quarters albumen. The patient had some hæmoptysis, the quantity of albumen in the urine increased, and he sank delirious. Autopsy: The heart weighed about seventeen ounces. It was much hypertrophied, especially the right side. The left

ventricle was also thick, but not to anything like the extent of the right side, but its cavity was considerably dilated. The endocardium was throughout white and thick. The edges of the aortic valves were thickened so as to cause much incompetence, and the mitral valve was thick and rather small, only admitting two fingers. The aorta began to contract opposite the left carotid artery, and then narrowed. Just beyond the closed orifice of the ductus arteriosus it was closely constricted as by a cord, and the lumen of the vessel would only allow of a No. 6 or 8 gum elastic catheter being passed through it. Beyond this the artery again dilated to its full size, though towards the end of the thoracic aorta it was diminished. The abdominal aorta and the iliac arteries were also rather small. The ascending aorta was "full large," but healthy. Beyond the constriction the coats of the vessel were thin and on the concave aspect white and cicatricial looking. The great vessels were all thick and large. The superior intercostal and internal mammary arteries were about twice the normal size, the former being especially tortuous. The epigastric arteries were not particularly large. The ductus arteriosus was closed, and there was a good deal of fibrous tissue around it. The liver was nutmegged, and the kidneys congested and tough.

CASE 9 (*see Insp.*, 1858, No. 161, and *Trans. Pathological Soc.*, vol. x., 1859).—James D., æt. 22, was admitted under Dr. Rees. He had had good health until puberty but after that time was only able to do light work at the docks. Later on he joined the militia but had to leave the service as the work was too severe for him. He then again went to the docks and continued at his work until two weeks before admission to the hospital and six weeks before his death. On admission, he was very dyspnoic and the legs were cedematous. The heart was enlarged, and its action tumultuous. No bruit was heard. He was thought to be suffering from cardiac disease, although its nature was not very clear, but it was said to be more like disease of the arch of the aorta. These symptoms increased until death. Autopsy: The heart was much enlarged but its normal shape maintained, the hypertrophy of the right side being proportional to that of the left. Two of the aortic valves had coalesced. The mitral curtains and cords were malformed. The right papillary muscle was scarcely present and the cords spread out. On the left side the muscle column was wanting. At the intermembranous septum a pouch projected into the right ventricle. The arch of the aorta was of the usual size but then gradually narrowed, having a conical shape. Immediately beneath the left subclavian artery the aorta became constricted, so as only to allow the passage of an ordinary probe, the opening being rounded and having slightly projecting edges. Below this the aorta was rather small and its wall thinner than usual, and the iliac arteries also, but after receiving the epigastric branches they became suddenly larger. The large trunks from the arch were all larger than normal, and the vessels from the subclavian much increased in size. The thoracic vessels, the superior intercostal, and the intercostal arteries lower down were all much enlarged.

CASE 10 (*see Trans. Pathological Soc.*, vol. i., 1847).—The patient, a female, æt. 34, was admitted in January, 1847, under Dr. Barlow. She was of phlegmatic temperament and stunted development. The face was pale, puffy, and abundantly streaked with capillary veins. At six or seven years of age she had suffered from palpitation, shortness of breath, occasional sickness

and swelling of the legs and ankles. She was subjected to cough and expectorated a good deal, the sputa being occasionally streaked with blood. At fourteen years of age the patient went to live in the country, and after that remained in apparently good health until twenty years of age, when the previous symptoms recurred. In 1846 the patient was in Guy's under Dr. Addison for the same symptoms and went out in three weeks relieved, but since her discharge she had gradually become worse again. On admission, the symptoms present were of the same nature as those above-mentioned. The chest was narrow and very prominent. There was oedema of the legs and ankles. The heart's action was forcible and irregular. Two loud "sawing" murmurs were audible over the præcordial area, most marked over the third and fourth sterno-costal articulation on either side. The patient became gradually weaker and died three months after admission. She was said to have been a seven months' child. Mr. Wilkinson King had diagnosed the presence of a patent ductus arteriosus. Autopsy: The heart was enlarged and, with the blood-vessels attached to it, weighed eighteen ounces. Both ventricles were dilated and the walls thickened. The aortic orifice was stenosed measuring two and six-tenths inches. Large, bony and cretaceous vegetations, the size of a filbert nut, were attached to one of the anterior aortic cusps, and there was an aneurysmal bulging of this valve. The adjacent cusps were perforated, and there were fibrinous vegetations on one of them, and also a few small vegetations on the mitral valve. Within the aorta, a quarter of an inch above the valves a miniature fourth valve was present. The aorta was narrow throughout, and there was much atheroma everywhere. Immediately opposite, or perhaps slightly above, the origin of the left subclavian artery there was a tight narrow band, more than one-third the circumference of the vessel, very much narrowing its calibre. Immediately beyond this band was a circular opening, the remains of the ductus arteriosus, surrounded by small vegetations, leading into the pulmonary artery.

CASE 11 (*see Insp.*, vol. 34, p. 7).—Martha W., æt. 17, was admitted in 1845 under Dr. Babington. The history of the case states that about three weeks before her death she had fainted in chapel, and during the day retched several times. The retching continued for some days, and was associated with vomiting and pain in the head. Later she passed into a dejected condition and remained speechless until her death, which took place about half an hour after a fainting fit accompanied by vomiting, similar to that above described. She was much emaciated. Autopsy: The report states that the heart was "almost a man's," the left valves being "coarse." The arch of the aorta was "smallish and strong, narrowing much and contracting to a probe fissure, then at once fairly dilating." The ductus arteriosus admitted a bristle. The mammary epigastric and first intercostal arteries were very large and thin. Some blood was extravasated over the anterior half of the brain, with a layer of clot below. There was a hæmorrhage in the anterior end of each hemisphere, the right, the size of a walnut, the left, the larger, had ruptured into the ventricle.

CASE 12 (*see Insp.*, vol. 35, p. 12).—William T., æt. 26, was admitted in 1847 under Dr. Rees. There is no clinical account of the case in the post-mortem records. Autopsy: The pericardium was adherent everywhere, but

although the adhesions were of old standing, yet they were easily broken down with the finger. The heart was hypertrophied and dilated. The aortic valves were opaque, thick, and the edges rounded and fissured. The mitral valve was very thick, the edges being rounded, and the chordæ tendinæe enlarged, opaque and rigid. The tricuspid valve was thicker and more opaque than usual. Four vessels arose from the aortic arch, viz. (1), the right carotid; (2) the left carotid; (3) the left subclavian; (4) the right subclavian, which after coming off from the posterior portion of the descending part of the arch crossed the vertebral column behind the œsophagus and trachea, and so on to its destination. At the point of origin of the right subclavian artery the aorta presented internally a ridge, and was also slightly contracted. The arch of the aorta was a little dilated, but below the ridge it appeared of smaller diameter than usual. There was œdema of the lower extremities and scrotum, and blood-tinged serum in the abdominal cavity.

CASE 13 (*see Insp.*, vol. 33, p. 16).—A woman, whose age is not stated, was admitted in 1844. There is no clinical history of the case. Autopsy: "The extremities of both hands and feet were of a leaden hue, suggesting the blue disease." The skeleton was deformed and the spine slightly curved. The heart occupied a median position in the chest, the right side, which was exceedingly dilated and hypertrophied, forming the apex and extending about an inch beyond the left ventricle. The left side of the heart was small. Two venæ cavæ entered as usual the right auricle, and also all the right pulmonary veins. The foramen ovale was patent, and posterior to it the septum was deficient over an area the size of half-a-crown. The aorta was narrow, and its valves small and thin. A well-marked contraction was present to the left side of the left subclavian artery, and below that the aorta was again somewhat increased in size, but still unusually small and gradually diminishing down to the common iliac arteries. There was some atheroma in places. Neither the internal mammary nor intercostal arteries presented any obvious enlargement. The pulmonary artery, its aperture and branches were enormously dilated.

CASE 14 (*see Insp.*, vol. 32, p. 45).—Eliza W., æt. 40, was admitted in 1842 under Mr. Cooper. There is no clinical history of the case in the post-mortem report. Autopsy: A saccular aneurysm, the size of a small adult heart, was found springing from the ascending aorta on the right side. It had caused some erosion of the ribs. The aorta was dilated to the end of the arch, where there was "a slight narrowing beyond the pit of the duct, a partial thickish ridge." Just beyond there was a dilatation. The aortic valves were a little contracted, the right ventricle was rather large and thick, and the pulmonary artery large.

CASE 15 (*see Insp.*, vol. 31, p. 111).—Stephen S., æt. 21, was admitted in 1841 under Dr. Addison. There is no clinical history of the case in the post-mortem report. Autopsy: The left auricle is described as being wider than the right and "thick and opaque within." The left ventricle was "very large and thick," and the mitral valve a little opaque. The aortic orifice presented only two valves and was wide. The ductus arteriosus was patent and wider than a goose quill, and immediately to the right of it the aorta was contracted to less than half an inch in diameter. Just beyond this on

the convexity was an incipient aneurysm, a circular patch as large as a penny, of inflammatory softening and yielding of all the coats. The anterior part of the falx cerebri was deficient. A large artery ran from the middle of the basilar artery into the right cavernous sinus.

CASE 16 (*see Insp.*, vol. 19, p. 78).—John B., æt. 33, was admitted in 1834 under Dr. Back. He was a big stout man, and was said to have been ill about three months. The history of his case states that he came into the hospital with manifest over-action of the heart and serious symptoms of chest obstruction. The bruit de scie was extreme. The patient quickly sank and died in a comatose condition. Autopsy: The legs were slightly cedematous. The heart was quite twice the natural size, all its cavities seeming equally affected, being dilated and hypertrophied. The pulmonary artery was very wide. All the valves were probably incompetent from widening of the cavities. The aorta was wide in its ascending portion, and there was a slight constriction near the former opening of the ductus arteriosus. (No further particulars are recorded in the post-mortem report.)

CASE 17 (*see Insp.*, vol. 18, p. 19).—James H., æt. 38, was admitted in 1834 under Mr. Key, suffering from jaundice due to an impacted gall-stone. Autopsy: The body was that of a moderately fat man and was slightly jaundiced. The heart was of good size, the left ventricle being somewhat hypertrophied. The mitral and aortic valves were thickened. There was a sudden annular constriction of the aorta of little more than a line in breadth, and situated about one inch beyond the left subclavian artery. The contracted orifice would not admit the little finger; it was somewhat indurated, and external to it were some large and indurated glands. The aorta was narrow and thin above the constriction, and below thickened and atheromatous. The abdominal aorta and the iliac arteries were of natural size; the intercostal arteries were large.

CASE 18 (*see Insp.*, vol. 15, p. 1, and Drawing 41 (12), also Monthly Journal of Medical Science, 1844, vol. iv., p. 32).—The description of the specimen was written in 1839 by Mr. Wilkinson King. It came from a child æt. $2\frac{2}{3}$ years, who was said to have always been thin and to have never been able to walk. The child died suddenly. Description of specimen: The aorta and pulmonary artery were transposed, and the septum of auricles and ventricles perforated. The right auricle was dilated and hypertrophied, and the fossa ovalis large and cribriform, having apertures in it the size of a common black lead pencil. The right ventricle was large and strong. The left auricle was of moderate size and received only two veins. The left ventricle was small. The ductus arteriosus was closed, but beyond this the aorta appeared decidedly smallish.

TRAUMATIC SUBDURAL HÆMORRHAGE.

AN ATTEMPT AT A SYSTEMATIC STUDY
BASED ON THE EXAMINATION OF SEVENTY-
TWO COLLECTED CASES.

By W. H. BOWEN, M.S.

My attention was first directed to the subject of extravasation of blood beneath the dura mater in the latter part of the year 1902, when as house-surgeon to Mr. C. J. Symonds, I worked under that surgeon, and had the care of his beds in his absence. The case which I shall give in full below, was admitted soon after I first took on the appointment of house-surgeon, and it was whilst endeavouring to find out some description of the signs, symptoms, etc., of the condition, that I discovered how little systematic study of the subject had been made. Unable at that time to go further into the matter, I left the subject, but towards the latter half of the year 1903, having time to spare, I thought it could be spent at least profitably in looking up the literature on the subject. Mr. Jacobson's well-known paper on Middle Meningeal Hæmorrhage, has put cerebral compression by blood-clot in almost all its aspects, whilst the numerous papers and lectures on injuries to the brain and skull have made a heterogeneous collection of cases unnecessary. The following are therefore a few points in favour of considering the form of head injury about which I write. In the first place, I may

say that in the English language I could find no systematic consideration of compression of the brain by blood which is situated beneath the dura mater and yet is not intracerebral, and even in the few cases in which reference is made to the subject, and conclusions drawn, especially with regard to symptoms and diagnosis, these conclusions, having been based upon but a few cases (one case alone being usually given), are, if not erroneous, at least unreliable. Again, a systematic study of the subject, based on a large number of cases, has brought into prominence one or two points which are scarcely recognised, and certainly not emphasised in text-books, one being the very long lucid interval which may be present, another that consciousness may never be lost, and a third, into which I shall go more fully when dealing with treatment, that the blood may be mixed with a watery fluid which is either serum or cerebro-spinal fluid. As an additional reason for this paper, I may say that the detailed study of the signs and symptoms in a large number of cases of traumatic cerebral compression, in the way that I have arranged them in this paper, has seldom been made; whilst the frequency with which the diagnosis has been an open question or wrong, would seem to show that the subject is one that would repay investigation.

I am greatly indebted to Mr. Symonds for permission to report the following case. It was the study of this case which first aroused my interest in the subject, and led to those investigations with which this paper deals. The report is a copy of notes I made at the time:—

“J. J., is a man said to be 40 years of age, but looking older, a seaman and a Swede, but speaking English well. On October 10th, 1902, whilst working in the Surrey Commercial Docks on his ship he fell down the hatch, was picked up unconscious, and brought up by a dock policeman who knew nothing about him, and, his ship sailing next day, his friends were never seen.

“When admitted, he was unconscious, with rather stertorous breathing. He had been in this condition since the accident. There had been no sickness, no hæmorrhage from ears, nose, or mouth. Two scalp wounds were present, one on the right side

of the middle line of the head, over the right parietal bone running obliquely forwards and outwards, and two and a half inches long, the lower is three inches long and is directed more transversely just below the occipital protuberance. The upper one shows that the occipito-frontalis is not torn, the lower has a small area of bare bone exposed. The edges of the wound are clean cut. There is very little hæmorrhage. The wounds were most thoroughly scrubbed with an antiseptic lotion, the head being as fixed as possible. The patient was sufficiently roused by this cleansing to endeavour to resist and muttered to himself. A little iodoform was sprinkled on the wound. Iodoform gauze was applied with blue wool, and the patient at once sent into the ward. An ice-bag was applied to his head and Calomel grs. 5 was given. He recovered somewhat a few hours after he came in. His stertorous breathing gave way to normal respirations. His pupils were from the first equal and reacted to light. He had no sign of paralysis.

"Left quiet, with light nourishment (first, milk for about two days, then farinaceous diet, and later fish) he made apparently a very good recovery, his head being less painful day by day. There can, however, be very little doubt, looking back upon his recovery, that it was far from perfect. No special feature can perhaps be pointed to as marking him out as still in danger, but there remained an air of languor, a feebleness in movement and a failure to respond quickly, which on retrospection we are bound to consider as due to persistence of some cerebral lesion. It is perhaps with greater emphasis that we can lay stress upon this point, when we remember that he was a seaman, used probably to climbing rigging and to quickly obeying orders.

"On October 21st, the patient was out in the open air and apparently recovered; he smoked his pipe, and conversed with his fellows, but there was always the indefinite feebleness. On the few occasions on which his eyes were examined, the pupils were equal and reacted to light. He had no paralysis.

"On October 23rd, he should have gone out to a convalescent home, but as there was a little bare bone at the bottom of the

lower wound which had not quite healed, it was decided to let him remain in another week.

"On October 25th, the patient seemed to be ill; he had a severe headache, did not take his food, seemed inclined to sleep the whole time, and his pulse rate became slow. The wounds were carefully examined; the upper one had quite healed, the lower one was still open with a bit of bare bone at the bottom of it, but it was quite clean. The eyes showed the pupils to be equal and reacting to light and accommodation. There were no paresis or paralysis. Tongue a little furred; no sickness.

"On October 28th, as he seemed to be sinking into a condition of coma, it being very hard to rouse him at all, it was decided to trephine him over the area of bare bone. It was thought that an extradural abscess might be present. The centre of the area of bare bone was used for the pin of the trephine. Chloroform was given. The dura mater was found normal. It was opened, and a little serum escaped, otherwise all was normal. The opening in the dura mater was not stitched up, but the skin wound (an incision transverse to the original cut, thus making the wound crucial, was made) was sewn up with salmon gut and the patient put back to bed. The temperature had been normal the whole course of the illness. The operation made practically no difference to his condition, but during the next couple of days he developed paresis on the left side, with rigidity on the right. This affected the upper extremities much more than the lower. The change was gradual, the grip of the left hand growing less and less until ultimately it disappeared, so that whereas on shouting at him he would give a feeble grasp with the right hand, he gave none with the left, even on putting one's hand into his and squeezing it. The right upper limb in what appeared to be his position of rest occupied the position seen after hemiplegia, *i.e.*, the elbow flexed somewhat beyond the right angle, the forearm pronated, the wrist and fingers flexed, but he could and would move all his limbs at times, and at times one found him supporting his head with his right arm as he lay on his right side. He always lay on his right side with his head turned to the right. This was practically the condition found on

October 31st. The right pupil was a little larger than the left, and Dr. Eason, who saw him, stated that he had commencing optic neuritis on both sides. No sickness. He was taking food very badly. The coma was certainly deepening but he still put out his tongue a little if repeatedly shouted at. No facial paralysis. Mr. Symonds decided that he should be trephined on the right side of the skull over the temporal region. Chloroform was again administered. A flap was turned down and a circle of bone removed with a one inch trephine, dura mater exposed appearing bluish in colour. This was incised, and immediately some thick dark blood escaped, followed by a quantity of bloody serum. The condition within the dura mater was then examined, and it was found that over the whole of the right Rolandic and frontal regions, the brain was compressed by this fluid, which welled out under some pressure. The anæsthetist noted that after the fluid was let out the pupils reacted to light. They had not done so before operation whilst the anæsthetic was being administered. The patient was put very lightly under the anæsthetic. The flap was punctured at its centre, and a rubber drainage tube put through into the cavity left between the depressed brain and the dura mater. Dressing put over with plenty of blue wool. The pulse was 70 when the patient was removed to bed.

"The operation was about 4.30 p.m., at 10.30 p.m. the following was his condition: Pulse 64, regular; no sickness; right side being rigid and practically the same as before operation, left side very limp, and practically the same as before operation; pupils equal and react to light. Very dull and apathetic. Tongue put out on demand. Right plantar reflex flexor, left extensor. Knee-jerks present on both sides. Ankle clonus on both sides. He moves both legs.

"On November 1st, he was still very dull but certainly improved. Pulse 60 to 64. Both arms rather rigid, but right much more than left. Incapable of grasping with left hand. Had two slight convulsive attacks in the morning about 9.30 and what appeared to be an attack of syncope at 11 a.m. (pulse scarcely to be felt; loss of colour; pupils equal). The wound was dressed and the

trephine opening very gently irrigated with normal saline solution. There was not much discharge from the wound.

"On November 2nd, pupils equal and react to light. The temperature is down; no sickness, very drowsy still, puts tongue out when told to. White fur on dorsum of tongue. Pulse 64, regular; wound quite healthy. Rigidity of right arm, paresis of left. Capable of getting out of bed and he then uses all his limbs. The evacuations are passed under him (this has been so since October 25th), no facial paralysis.

"On November 3rd, when the patient was seen this morning he had his eyes open and was lying on the left side. He put his tongue out, and paresis seemed to have disappeared from left side. The grasp of either hand was feeble. Temperature normal, pulse 66. He had several convulsive twitchings of the mouth during the day. As they were only noticed when he lay on the right side, it was thought that they might be due to pressure from the drainage tube on the face area; with this idea the tube was shortened, but during the night he had three other attacks, and these were present when the patient was on his left side as well as on his right. He was restless in the evening and tried to get out of bed.

"On November 4th, he was sitting up in bed with eyes open when seen this morning, but he still has very imperfect muttering speech, which prevents one getting proper replies to questions. No sickness, temperature normal, feeble grasp with both hands. Pulse regular, 64. At 10 p.m. patient was quite quiet, pulse 80, temperature 98°, respiration 20. At 10.30 p.m. patient had a fit. There were violent twitchings of face and eyes, also left arm and leg, and slight frothing at mouth. The fit was followed by marked blowing in and out of cheeks. Its duration was two minutes. The patient was rather blue during the fit, but there was no sign of collapse afterwards. At 11 p.m. there was a similar fit, but slightly shorter in duration. At 12.20 on the 5th there was a similar fit, but more violent and longer and accompanied by slight twitchings of right arm. Pulse 80. At 1.15 a.m., 2.30 a.m., 3.30 a.m., 5 a.m. and 7 a.m. fits occurred. He had his last fit, a severe one, affecting the whole of the left

side of the body, later on during the morning of the 5th. Dr. Eason stated that the optic neuritis was more pronounced than when seen before.

"On November 6th there were no fits and no paralyses, but it was noticeable that the pulse was very irregular in rate and force. On the 7th he asked for everything he wanted, and words being as distinct as before the relapse. Said he had no pain and no headache. The tongue was moist and clean. The drainage tube which had been put down level with the dura mater was taken out to-day.

"On November 11th the pulse was regular as regards frequency but irregular in force.

"From this time the patient never went back, but his recovery was very slow. His movements were slow and hesitating for a long time, and he had no desire to converse, although he could speak quite well. On the other hand, he had no headache, took his food well, and had a normal pulse, temperature and respiration. When he was discharged to a convalescent home on January 8th, 1903, he was practically perfectly recovered in all respects, save for slightly delayed cerebration. He had memory for past events but not for recent ones connected with the accident and convalescence. When he returned from the convalescent home at the end of three weeks he was, as far as it was possible to say, quite recovered, and Dr. Eason, who examined his eyes, said it was impossible, from the condition then present, to say that optic neuritis had ever been present."

Such is the history of this case, and before passing on to a systematic study of subdural hæmorrhage, the result of injury, I would call attention more particularly to the long lucid interval, the absence of any signs of cerebral laceration, or of fractured skull, the difficulties in making a diagnosis, the correct conditions only being found at the second operation, the appearances found at the operation, especially the bluish colour of the dura mater, which enclosed blood clot and bloody fluid with marked compression of the brain, the presence of optic neuritis, and the delayed convalescence but ultimate recovery. These matters will each be considered more fully later.

I had originally intended to base this paper entirely upon the cases reported in the surgical reports of Guy's Hospital, kept in the surgical registrar's room, and with this end in view went carefully through these reports for thirty years, viz., 1870 to 1900, supplementing them wherever possible by the post-mortem records, but for more than one reason I found that this was not sufficient. In the first place, the reports only supplied one case similar to the above, and ending in recovery, and in the second place, I quickly found many side problems came into the question. These required investigation, necessitating reference to many books, and it was whilst consulting these references that I first found some of the best and most typical examples of cases coming under class A, and led me finally to refer to that encyclopædia of medical reference, the Index Catalogue of the Surgeon-General's office of the United States Army. A bibliography will be given at the end of this paper, but I may say that I consulted the Transactions of the Medico-Chirurgical, the Pathological, the Clinical, and Medical Societies from their commencement up to the year 1902, the Reports of Guy's, St. Thomas's, St. Bartholomew's, Westminster, and King's College Hospitals, up to the year 1902, Brain, American Journal of Medical Sciences, the Journal of the American Medical Association, the Medical Record, and the Boston Medical and Surgical Journal, in addition to the many British medical journals and papers. By this means a total of seventy-two cases of subdural hæmorrhage, the result of injury, was found. These have been divided into two groups of cases under class A and class B; the former may be described as cases of practically pure compression by blood clot, there being no reason to suspect that laceration, or even contusion, were present in any of the cases; the latter, class B, on the other hand, have been grouped together because in these cases the compression had been complicated by the presence of laceration, very pronounced contusion or severe general concussion which has considerably influenced the course of the cases, and must, in most cases at least, be looked upon as the real cause of the fatal termination which followed. Extra-dural hæmorrhage is associated with the subdural hæmorrhage in

five cases (see under Condition of the Brain), all five coming under class B.

A few words may here be appropriately written upon the value of the word "subdural" as opposed to "subarachnoid" or "intermeningeal" or "subpial," etc. Phelps, in his work "Traumatic Injuries of the Brain," p. 44, says, "If the prefix epidural is invariably used to characterise a hæmorrhage which separates the dura from the cranial wall, "pial" to characterise a hæmorrhage into that membrane from rupture of its vessels, and "cortical" to characterise a hæmorrhage upon the surface of the brain from laceration of its substance, both the source and location of the hæmorrhage will be expressed in a single word with accuracy and conciseness, and the description of cases much shortened and facilitated." Despite this, however, and whilst not for one moment discussing its accuracy as far as it goes, I hold that the word "pial" does not sufficiently describe those cases including, I believe, the majority of cases of extensive hæmorrhage belonging to class A, in which the blood is situated in the so-called "cavity" of the arachnoid, *i.e.*, the subdural space. Again, cases are reported where blood is situated both supra- and sub-arachnoid and supra- and sub-pial (see cases 18, 25, 26 and 64), and also cases where the exact position and origin of the blood is difficult to make out. Hence I have adhered to the word subdural, as embracing all cases, and as being used commonly in reference to both suppuration and hæmorrhage beneath the dura mater. There is, of course, the objection to subdural that it includes intracerebral hæmorrhage, but at the same time the special term of cerebral hæmorrhage is so universally given to this form of hæmorrhage as cerebral abscess to intracerebral suppuration, that the expression subdural is invariably looked upon as also equivalent to extra cerebral.

It is my intention, before passing on to the signs and symptoms, diagnosis, prognosis and treatment of traumatic subdural hæmorrhage, to consider somewhat fully the etiology of subdural hæmorrhage, both traumatic and non-traumatic. It is important to consider the latter class of cases, often described as "hæmatoma of the dura mater," or "meningeal hæmorrhage," or

when of more remote occurrence and becoming organised, passing under the name of Pachymeningitis Interna Hæmorrhagica, because in these cases one expects to find those conditions present which tend to idiopathic hæmorrhage, and in which therefore but very slight injury would be sufficient to lead to extravasation of blood, whereas in a healthy body such an injury would have no such effect. The earliest consideration of this question that I found was Prescott Hewett's grouping in his paper read before the Medico-Chirurgical Society and published in vol. 28 (1845) of the Transactions of the Society "On extravasation of blood into the cavity of the Arachnoid." This writer divided the etiological factors into two groups, the first all "those leading to a decided determination of blood to the head by whatsoever cause produced," and the second those leading to and associated with arterial degeneration. Two other classifications of the causes of subdural hæmorrhage were found, one in the Journal of the American Medical Association for January 26th, 1901, where the subject is considered in an editorial article, and also the case No. 67 reported (class B) and the other which really deals with the etiology of Pachymeningitis Interna Hæmorrhagica in the Gazette des Hôpitaux, Saturday, March 13th, 1897. "Les Méningites Cerebrales Hæmorrhagiques," by Dr. F. de Grandmaison.

From these three sources and the many cases of idiopathic and traumatic cases singly reported in the Journals and Transactions, I have come to the conclusion that the following is the best classification which I can make.

A. CONDITIONS LEADING TO A DECIDED DETERMINATION OF BLOOD TO THE HEAD BY WHATSOEVER CAUSE PRODUCED (P. Hewett).

This group included—

1. ALCOHOLISM.

Grandmaison (*vide supra*) points out that alcohol predisposes in two ways:—

1. By its sclerosing property.
2. By the congestion it brings about.

With regard to the former, I would call attention to the case reported by Dr. R. Van Santvoord, and referred to when dealing with prognosis (p. 114).

The latter is important, since despite the proverb that "Providence watches over a drunken man," hospital experience shows that he is particularly liable to injury, and the injury being very often to the skull acts indirectly upon a congested brain.

I have classified the seventy-two cases with a view to their bearing upon this subject, and the following is the result:—

In class A, out of the thirty-six cases reported, there is some alcoholic history in nine, and of these five died. Of these nine cases, in only three is it definitely stated that the man was drunk when the accident occurred, and of these three, two recovered and one died. In only one case does it say that the man was a total abstainer, and he died (case No. 3).

In class B (also thirty-six cases) there was some alcoholic history present in eight cases, and of these seven died. In the other twenty-eight cases, there is no record of any history of alcoholism, and no post-mortem evidence, if a post-mortem was made. Of the eight cases in which an alcoholic history was present, in three the patient was undoubtedly drunk when the accident happened, and in two he was probably drunk. In all these cases the patients died.

It is scarcely necessary to refer to the unsatisfactory nature of the above evidence, the absence of negative history in the reports is noteworthy in the majority of cases, and the known difficulty in getting a reliable history from hospital patients, makes a negative history often of doubtful value when obtained. The absence of any report upon the condition of the liver in the post-mortem examinations made in cases coming under class B, also nullifies the value of the cases when considering this question.

2. ACUTE DISEASES.

This class of case is placed here owing to the liability to cerebral hyperæmia. Cases of the occurrence of subdural hæmorrhage with anthrax are reported (*British Medical Journal*,

July 20th, 1901, and Soc. Med. des Hosp., 2 Mars, 1894), and with erysipelas (M. Lancereaux referred to by Grandmaison). These cases have no bearing on traumatic subdural hæmorrhage, and are in the form of diffuse hæmorrhage (thin layer) in the subarachnoid space.

3. GREAT ANXIETY OF MIND (Prescott Hewett).

I find no case reported, but in connection with this class of case, the following case is interesting. It is reported in the *Gazette des Hospitaux* (Paris), 1884, lvii., p. 531, by M. Maheut, under the title of "*Hæmorrhagie dans les Meninges Cerebrales chez une jeune fille de treize ans.*" The case briefly stated is as follows:—

A girl, thirteen years of age, witnessing a quarrel between her mother and father (April 21st) was extremely frightened. The same night she was agitated, and vomited. Next day at 10 a.m. she lost consciousness, convulsions (convulsions eclamptiques) followed. She was taken to the Hôtel Dieu. Later she had general paralysis, pupils dilated, and not reacting to light. Pulse 120. Temperature subnormal. Ice was applied to the shaved head, calomel given, and artificial feeding resorted to. She died April 27th–28th at night, remaining practically the same until the end.

Post-mortem.—On opening the skull the dura mater was seen to be slightly adherent over the convex part of the hemisphere, with a tinge of red beneath it. Beneath this membrane was a coagulum thick enough to conceal the cerebral convolutions. The deposit of blood was between the arachnoid and pia mater from the anterior extremity to the junction of the middle and posterior thirds. The blood was adherent to the membranes and difficult to free. The pia mater was intimately attached to the superficial parts of the brain, which showed on section a most pronouncedly injected state. The right hemisphere was feebly injected. The ventricles contained much yellow serous fluid without a trace of blood. The other organs were normal.

The case is extremely interesting on account of the age of the patient, the absence of any visceral lesions, and the cause which

led to the cerebral hyperæmia, and it would seem to attach additional significance to the importance of quiet and freedom of worry during convalescence and even subsequent lifetime of those who have once suffered from subdural hæmorrhage.

4.

Another interesting case which is not without a significance of its own is recorded in the post-mortem reports of Guy's Hospital (No. 230), for the year 1872. The patient, in the Eye wards under Mr. Bader, was being anæsthetised preliminary to operation. He struggled violently, became blue, the pulse ceased, and he died. At the subsequent post-mortem examination the sinuses were full of dark blood. There was blood on the surfaces of both hemispheres into the pia mater, and the vessels of the membrane were also very turgid. The brain weighed fifty-two ounces; puncta vasculosa were dark and very evident, otherwise normal. Internally viscera congested, otherwise normal.

The significance of such a case when the operative treatment is considered becomes obvious. It is noticeable that chloroform alone was used and that there is no mention of any visceral lesion. It is also to be remembered that many cases of traumatic subdural hæmorrhage, especially those coming under class A, are operated on during the lucid interval.

5. THROMBOSIS, OR OBSTRUCTION OF THE LONGITUDINAL SINUS.

As regards thrombosis, under the title of "Cases from Sir Dyce Duckworth's Wards," by F. E. A. Colby, published in vol. xxviii. of the St. Bartholomew's Reports, is a case of thrombosis of the superior longitudinal sinus. At the post-mortem examination congenital heart disease was found, with thrombosis of the superior longitudinal sinus, rupture of a vein of the pia mater from distension, and subarachnoid hæmorrhage.

In reference to obstruction of the superior longitudinal sinus, I may refer those interested in the subject to the New York Medical Journal for January 24th, 1885, p. 104, where the question of meningeal hæmorrhage in the new-born, associated with difficult labour, is discussed, and to the *Semaine Medicale*,

1890, 10, p. 408, where M. Kundrat explains hæmorrhages in newly-born children as being due to the displacement of the parietal bones during labour, compression of the longitudinal sinus, regurgitation of blood towards the veins of the convexity and rupture of these veins. On the other hand, Cushing, in the Mütter Lecture for 1901, entitled, "Some experimental and Clinical observations concerning states of increased intracranial tension," the general methods of the experiments performed and conclusions which they led to, being published in the American Journal of Medical Sciences for September, 1902, states that the arrangements of the veins opening into the superior longitudinal sinus allows of a valve-like action preventing regurgitation of blood from the sinus into the veins. It is proved by the fact that injection into the sinus will not pass back into the veins. His experiments were made on dogs, and he also states that "contrary to the general belief, the longitudinal sinus, in the dog at any rate, may be completely collapsed by an increase of intracranial tension. With reference to the effects of compression, the following sentences are important. "If the distension of the bag is further increased the intracranial tension in its vicinity finally reaches that of capillary pressure, at which time the convolutions of the brain, not only in the neighbourhood of the foreign body, but if the process is still further advanced, even in remote parts of the hemisphere abruptly lose their rosy colour and become blanched, the veins meanwhile remain filled with dark blood, whose means of escape seems to have been cut off, as it were, at both ends. Now, such a condition of anæmia of the hemispheres may be brought about, in the dog at all events, with but little evidence of the so-called major symptoms of compression." Hence it appears that although regurgitation does not take place from the sinus, yet the blood is unable to pass from the pial veins into the sinus; engorgement results, and should the vein be torn, the venous congestion would lead to excessive extravasation of blood; the intracranial tension is further raised, and in fact a vicious circle is formed.

In discussing the origin of the blood, however, it will be found that I have arrived at the conclusion that a venous origin must

be very rare, owing to the fact that compression means forcing the blood from the brain, which is almost synonymous to a greater pressure of blood in the veins than in the arteries, if the source be looked upon as venous. This is impossible as far as we know, but that some other factor is introduced is shown by case 20, where there is every reason to believe that the lateral sinus was the only source of blood, and yet compression was present and the description of the post-mortem examination says that there were several ounces of blood in the middle fossa. The cases recorded of extradural hæmorrhage, from rupture of the lateral sinus, point in the same direction. It almost looks as though clinical experience and experimental pathology were at variance.

6. POISONING BY OPIUM (Prescott Hewett).

7. ACTIVITY IN OVER-HEATED ROOM (Journ. Amer. Med. Assoc.).

I can find no cases reported of either of these conditions. Prescott Hewett states that he has seen subdural hæmorrhage due to the former, and looking upon it as probably due to venous congestion secondary to respiratory failure, I have put it here.

B. CONDITIONS ASSOCIATED WITH ATROPHY OF THE BRAIN.

This group of cases included (*a*) the cases of subdural hæmorrhage occurring in the insane, the hæmorrhage being secondary to loss of support of the vessels owing to cerebral atrophy. In the Transactions of the Pathological Society, vol. xlv., is a paper entitled, "Some gross lesions in the Brains of Lunatics, with remarks upon the frequency with which the two sides of the brain are affected," by Cecil F. Beadles. The writer gives a table showing the vascular lesions met with in three thousand three hundred consecutive brains examined. In the Journal of Mental Science for January, 1888, is a paper entitled "On Hæmorrhages and False Membranes within the cerebral Subdural space occurring in the insane (including the so-called

Pachy-Meningitis)," by Joseph Wigglesworth, M.D. Lond. The paper is based on four hundred unselected post-mortem examinations. Dr. Wigglesworth's conclusions are given on p. 523 of that Journal, that coming under No. 5 is as follows: "Whilst in the great majority of cases traumatism may be confidently excluded, there seems reason for believing that under favourable predisposing conditions a slight injury may start a hæmorrhage which may prove fatal." In the seventy-two cases reported at the end of this paper there is no reason to suppose that any primary degenerative change in the brain existed in any of them. It is noticeable, however, that in the case reported by Taylor and Ballance (*Lancet*, August 29th, 1903) to which I shall refer when discussing prognosis, the family history shows a clear tendency to mental instability; the mother committed suicide, one sister was in an asylum, and an aunt had been temporarily insane.

b. The cases associated with wasting diseases, such as phthisis, have no bearing on our subject beyond the possibility that patients suffering from such conditions are more liable to subdural hæmorrhage from slight violence.

C. THOSE CONDITIONS LEADING TO, OR ASSOCIATED WITH, DEGENERATIVE CHANGES IN THE BLOOD VESSELS.

a. Atheroma and aneurysm.

Subdural hæmorrhage due to rupture of aneurysms is not at all uncommon. Two cases are reported in vol. xxviii. of the St. Bartholomew's Hospital Reports in the paper to which I have made reference before. In these cases the aneurysms involved in the one case the left sylvian artery, in the other the anterior communicating artery. In the post-mortem reports of Guy's Hospital for the year 1870 (No. 23) there is recorded subdural hæmorrhage due to ruptured aneurysm of the middle cerebral artery in a man who was brought in dead. That investigation would reveal many other similar cases I have no doubt.

With reference to the association of subdural hæmorrhage and atheroma, it would seem right to conclude that in the presence of the latter comparatively slight violence would lead to the former. Now, if this were so, we might expect more particularly those cases coming under class A to suffer from this form of arterial degeneration, for there can be little doubt, from a consideration of the two groups of cases, that whereas those coming under class B were due in most cases to the severest forms of violence, accompanied by fractures of the skull and severe injury to the brain, those coming under class A were the result of slighter violence if we take into consideration the absence of fractures or cerebral injury. Unfortunately, no records have been made of the condition of the vessels as regards tension or other points which might have thrown light upon the matter in those cases which recovered, numbering twenty-two out of the thirty-six collected.

Of the fourteen cases which died the following is an abstract:—

The condition of the vessels is mentioned in nine. In two cases they were "healthy" and "no signs of atheroma." These were in patients aged eighteen and forty-three respectively (cases 12 and 5). In case 12 the injury was a blow on the head whilst fighting. In case 5 it was a severe blow from the hook on the pulley of a crane.

In one the arch of the aorta was atheromatous and dilated, the patient was sixty years of age and the kidneys were cystic and granular (case 15). The patient fell twelve feet, pitching on his head.

In two cases there was slight atheroma in the commencement of the aorta. The ages of these patients were thirty-one and twenty respectively. In the former the kidneys were affected (case 16); in the latter they were normal (case 17). In the former (case 16) the accident was that the patient fell whilst walking, striking his head against the kerb, but he had a lucid interval of twelve days. In case 17 the man received blows whilst boxing, none of which were particularly hard. He passed into a state of coma at once, and died two hours after admission.

In one case the aortic valves were very atheromatous (case 31). The patient was aged 80. The accident was a fall downstairs, but it is only right to mention that the woman felt giddy before she fell.

The other three cases are Nos. 4, 13 and 19. In case 4, the vessels of the base of the brain "have some thickening and are opaque." Kidneys affected; age forty-eight. The woman was said to have fallen whilst drunk. In case 13, the subependymal vessels were fatty here and there; age thirty-five. This man was struck twice on the head by another man and died almost immediately after the blows. In case 19, the "walls of the blood-vessels of the brain seem degenerated." The man was aged forty-three. He suffered from old syphilis and was a hard drinker. He fell off a waggon, striking his head against the kerb.

Hence, in nine cases in which the vessels are mentioned, in seven they were diseased to a greater or lesser extent, and although no very decided opinion is to be ventured on such a small number of cases, it may be stated that the evidence points to a greater predisposition to extravasation in those the subject of arterial decay. At the same time, attention may be called to cases 18 and 36, where the hæmorrhage occurred in young boys as a result of comparatively slight violence. A comparison with the occurrence of idiopathic cerebral hæmorrhage, however, reveals that even here there are marked exceptions to the explanation of their origin in arterial decay. The three following cases will show the weakness of dogmatising. In the *Lancet*, for December 22nd, 1883, p. 1083, Dr. Walter Fergus records a case of death from cerebral hæmorrhage in a boy of sixteen years. In the *British Medical Journal* for 1897, Vol. i., p. 834, there is a report of a meeting of the Pathological Society of Manchester, at which Dr. Lea showed the brain from a girl twelve years of age, who died of idiopathic cerebral hæmorrhage. "The family history was good, no history of syphilis, no disease of viscera found, and the vessels were healthy. It may have been possibly due to rupture of a vein of the choroid plexus." And again, in the *British Medical Journal* for 1889, Vol. ii., p.

719, Dr. William Collier describes a case of death from cerebral hæmorrhage in a girl six and a half years of age.

At this point it will not be inappropriate to discuss the age of the patients, since if atheroma be at all a strong factor in the etiology of traumatic subdural hæmorrhage on the line of argument laid down above, it would rather be expected that the average age of the patients belonging to class A would approach that at which arterial decay begins to become common, viz., over fifty years of age, whereas the cases coming under class B, one would expect the average age to be under fifty, since such accidents would be most likely to occur in adult men in active employment. The following are the facts:—

Class A.

The youngest patient was one and a half years (case 29).

The oldest patient was eighty years (case 62).

The average age calculated from the thirty cases in which the age is given is thirty-nine years ten months.

In six cases no record of the age is given, but of these, all were adults save one, which was a boy.

Of the thirty cases in which the age is mentioned, there were—

From 1 to 10 years of age ...	2 cases.
" 11 " 20 " ...	3 "
" 21 " 30 " ...	5 "
" 31 " 40 " ...	6 "
" 41 " 50 " ...	4 "
" 51 " 60 " ...	6 "
" 61 " 70 " ...	3 "
" 71 " 80 " ...	1 "

(The numbers in age are inclusive.)

Class B.

The average age calculated from the thirty-three cases in which age is given is—

37 years and 8 months.

The youngest is eighteen (case 59).

The oldest is sixty-four (case 12).

It will be noticed that cases coming under class B bear out the argument that they should occur in men under fifty years of age. The average age calculated from the cases is that of activity and hard work. On the other hand, although the average of patients in class A is above that of class B, yet it is so slight as to be practically negligible, and is also the age of activity and hard work as opposed to that of arterial decay. Thus, the consideration of the age alone points to arterial degeneration playing no part in the occurrence of this form of injury.

The following considerations, however, have led me to retain my opinion on arterial decay as a predisposing factor:—

Firstly, the amount of evidence put forward when considering the presence of arterial decay in the cases coming under class A.

Secondly, the fact that people over fifty years of age are less liable to injury than those under fifty. Thus, of the thirty-three cases belonging to class B only six were over fifty years of age. These belong to the groups of cases in which the accident is received during the time they are employed in actively following some occupation.

And thirdly, that the cases belonging to class A, are taken from those reported in various Medical Journals, the majority of which are recoveries (twenty-two out of thirty-six), and as the prognosis for such conditions is better the younger the patient (speaking broadly, and referring chiefly to the point of above or below fifty years of age) the conclusions derived from them may not be an index of the true facts when taken alone.

b. Arterio-Sclerosis.—I have taken the condition found in the kidney at post-mortem examinations as an indication of the presence of any general degeneration of the arterial system. The few results of the examination of the urine which have been made I have considered under the Signs and Symptoms (q.v.). The following are the facts which have been gathered on the present subject:—

Class A.—Of the cases which died.

In one there was no post-mortem examination, and in one the brain only was examined; of the other twelve cases, the kidneys were abnormal in three, viz., case 4, where the right kidney was

atrophied and contained two cysts; the left was hypertrophied, weighing twelve ounces, also fatty and mottled; capsule adherent.

Case 15. Kidneys "cystic and granular."

Case 16. Large, and capsule in places adherent. Beyond swelling of the cortex, probably from slight infiltration, there was no evidence of disease.

It will be noticed that there was some arterial degeneration in all these cases.

The kidneys were normal in three cases, whilst their condition is not described in five cases. In the single remaining case, the viscera are described as "not healthy."

Class B.

Of the twenty-five cases in which a post-mortem examination was made, the kidneys are only referred to in seven cases.

In five cases the kidneys were normal, and in two cases abnormal; of the latter, in case 48, the left kidney was small and puckered, the right kidney was large with one scar. In case 55 the kidneys were granular.

It is impossible to draw any conclusions from the above data, but it is probable that the same significance may be attached to arterio-sclerosis as to atheroma. The systematic examination of all the viscera at post-mortem examinations made on cases of head injuries, especially such as come under class A, would throw more light upon the matter.

c. Escape of blood externally in case of intracerebral hæmorrhage (*Journ. of American Med. Assoc.*). Cases of this nature undoubtedly occur. Case No. 70 I take to belong to this group. The following case, admitted under Mr. Lucas, December 24th, 1889 (Report No. 380), is probably of the same class of case:—

H. C., aged 35, was riding on the box-seat of an omnibus when the latter collided with a cab. The patient fell off on to the pavement. On admission, he had profuse bleeding from the left ear, was unconscious but beginning to come round. The pupils reacted to light. There was slight bleeding from one nostril. No albumen or sugar in the urine. He was very sick soon after

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admission, later he became very violent, and was removed to the strong room, where he had to be strapped down. Temperature $100\cdot2^{\circ}$. On the 28th (four days after admission), he was quieter, complained of swimming sensations in the head, and also of pain which was chiefly on the right side of the head. Temperature $99\cdot6^{\circ}$, pulse 86, and respiration 30. Improvement followed, his pulse, temperature, and respiration being normal on December 31st, but on January 1st, he became decidedly worse. There was an internal squint of the left eye, left facial paralysis; when spoken to loudly he answers in a rational manner: on the 4th, he was much better again; on the 5th, he bit the end off the thermometer, swallowed the mercury, and was violently purged in consequence. On the 6th, pulse 120, temperature 103° , respiration 38. He died on the 8th, at 10 a.m., becoming very irritable, with rapid pulse and respiration. Temperature $103\cdot7^{\circ}$, and deviation of eyes to the left, pupil of right eye contracted, left dilated. At the post-mortem examination there was bruising of right frontal and temporo-sphenoidal lobes and subdural hæmorrhage over right side of the brain. In the right temporo-sphenoidal lobe there was a cavity containing semi-fluid blood, not communicating with the ventricle, but with the subdural collection of blood by a small aperture. There was no fractured base.

In the American Journal of Med. Sciences, vol. 113, there is reference to a case reported by Borsuk and Witzel in the *Archiv. für. Klin. Chir.*, 1897, Band liv. Heft I., of hæmorrhage into the white substance of the brain, followed by aphasia, hemiparesis, and Jacksonian epilepsy which was cured by surgical interference, and although there is no suggestion that the blood had broken through externally in this case, there is every reason to suppose that this would have happened.

The probability is that in these cases the layer of brain which lies between the area of hæmorrhage and the pia mater either gradually softens and so gives way, allowing the blood to escape externally, or as the result of some straining or other cause of rise of arterial tension, an additional extravasation of blood is able to break down this layer. It will be noted that in case 70

the patient was the subject of some form of chronic nephritis. We shall refer to the matter again under Prognosis.

D. TENDENCY TO HÆMORRHAGE ("Hæmorrhagic Diseases," *Journ. American Med. Assoc.*).

This has no bearing on traumatic, subdural hæmorrhage beyond the possibility of liability to hæmorrhage with slight injuries. The collected cases supply no evidence of such predisposing causes.¹

E. TRAUMA.

The etiological factors arranged under A, B, C and D are merely predisposing causes if they act at all, for this paper only attempts to deal with injury as the cause of subdural hæmorrhage, and beyond the importance of recognisable lesions being present and leading to spontaneous extravasation and, as stated at the commencement, being therefore liable to predispose to rupture of vessels if slight violence be applied, no further consideration of non-traumatic subdural hæmorrhage will be given. In passing on to a systematic study of subdural hæmorrhage based on the cases reported at the end of this paper, it is well that I should refer to two classes of cases which I shall leave unconsidered, since in neither can death be in any way attributed to compression of the brain, although in both a varying amount of subdural hæmorrhage is present. These two groups are, firstly, those cases where usually from a fall on the occiput or vertex, laceration of the frontal or temporo-sphenoidal lobes, or both together, follows, associated with some hæmorrhage from vessels of the injured brain. No compression symptoms are present, and the blood extravasated has no bearing upon the prognosis or treatment. It is this last sentence which definitely excludes them from class B. These cases are clinically identical with cases of laceration of the brain. The second group of cases

¹ Cases of Spontaneous Hæmorrhage associated with Anæmia, are quoted by Sir Samuel Wilks (*Guy's Hospital Reports*, 3 s., vol. v.), by Dr. W. P. Herringham in vol. xlv. of *Pathological Society's Transactions* (Splenic Anæmia), and by Dr. G. A. Sutherland in *Brain*, vol. xvii. (On Hæmatoma of the Dura Mater associated with Scurvy in Children).

are those of very severe concussion with a thin layer of blood overlying the brain at one or more parts. There is marked congestion of the whole of the brain substance with all the pathological signs of severe concussion. The patients clinically correspond to this condition and the blood extravasated is in a thin layer, causing practically no compression, although some signs of cerebral irritation may be present, or the blood may be in the form of a general infiltration of the subarachnoid space.

The following points will now be discussed :—

1. The nature of the injury.
2. The site of hæmorrhage with regard to the position of the head injury, *i.e.*, whether the extravasation of blood is below the seat of injury or opposite to it (*contre-coup*).
3. Presence or absence of fractured base, together with a consideration of the thickness of the skull in cases belonging to class A.
4. Condition of the brain.

1. THE NATURE OF THE INJURY.

It is difficult to say from a written description of an accident what its real severity was, but there can be no doubt that the violence of the injury in the cases belonging to class A was much less than that in class B. A reference below to the presence of fracture of the base of the skull and the presence of laceration or contusion, will also bear this out. At the same time it is doubtful whether in any of the reported cases the violence was as slight as in those cases of meningeal hæmorrhage reported by Erichsen and quoted by Mr. Jacobson. It is noticeable, however, that the slightest forms of violence recorded were much the same as in Erichsen's cases, where the patients slipped while going downstairs, striking the head against a wall.

In the simplest forms of violence coming under class A, the injury was in the form of blows received whilst boxing or in anger, and are to be found in cases 12, 13, 17 and 20. In case 12 the man was admitted conscious eleven days after the injury, and died the same night, apparently from a large secondary

hæmorrhage on the top of a small primary one. In case 13 the man was struck twice on the head, apparently in anger, and died immediately afterwards. The blows in cases 17 and 20 were received whilst boxing. In neither cases were the blows severe or fractures of the skull present, in both unconsciousness supervened almost immediately and persisted until death. The absence of any primary unconsciousness or external wound in many cases coming under class A also shows that comparatively slight violence may cause extravasation. In class B, as might be expected, the injuries were always severe.

2. THE SITE OF HÆMORRHAGE WITH REGARD TO THE POSITION OF THE HEAD INJURY, *i.e.*, WHETHER THE EXTRAVASATION OF BLOOD IS BELOW THE SEAT OF INJURY OR OPPOSITE TO IT (CONTRE-COUP).

The following is an analysis of the collected cases:—

	Class A.	Class B.
(a) Injury to one side of skull, hæmorrhage on the opposite side, <i>i.e.</i> , extravasation of blood by contre-coup.	4 cases.	8 cases.
(b) Injury not quite corresponding to opposite point, but for practical purposes the extravasation of blood is by contre-coup.	3 "	3 "
(c) Injury over occipital region, extravasation of blood over a motor region.	3 "	4 "
(d) Injury and extravasation definitely on the same side.	8 "	14 "
(e) No record to be obtained of relationship between injury and site of clot.	14 "	5 "

Class A.—Of the remaining four cases, in one the injury was on one side and the extravasation of blood was on both sides (case

34). In two, the extravasation was probably on the same side as the injury, and in the last case the lateral sinus was ruptured by a blow on the same side of the jaw.

Class B.—Of the two remaining cases:—In one there were injuries on both sides of the head but unilateral hæmorrhage, and in the other there was blood subdurally on both sides.

It will thus be seen that the extravasation of blood may be on the same side as the injury or on the opposite side. The fact that a blow on the occiput may lead to compression of a motor region by extravasated blood must also be noticed.

3. THE PRESENCE OF FRACTURED BASE WITH THE QUESTION OF THICKNESS OF THE SKULL.

Of the fourteen cases belonging to class A which died, fractured base was not present in a single one. Of the twenty-two which recovered, in two there were clinical signs of fractured base, viz., in cases Nos. 29 and 34. In the former there was bleeding from the right ear, and at operation the subdural hæmorrhage was on the left side and due to rupture of the middle meningeal artery. In the second case which is reported by Guthrie, it is said that the patient bled from the ears, eyes, nose, and mouth. The man had bilateral subdural hæmorrhage.

In class B, only six patients recovered, and of these one only (case 69) had signs of fractured base. In this case there was bleeding from the right ear. It occurred outside the hospital after the accident and also on the second day in the hospital. The patient, it is said, was unconscious for eight hours after the injury, and then had a lucid interval up to the time of operation. At the operation, no fracture of the skull was found, but there was some laceration of the brain.

Of the thirty cases which died:—There was evidence of fractured base during life or at the subsequent post-mortem examination in twenty cases (of these, there was external evidence, shown by blood from the ears, nose, or stomach, or subconjunctival hæmorrhage, in ten cases; of these the fractures were confirmed at a subsequent post-mortem examination in six,

whilst in four no post-mortem examination was made. There was no external evidence of fractured base but post-mortem showed it to be present in ten cases). There was no evidence of fractured base during life, and no post-mortem examination was made in one case. There was no evidence of fractured base during life, and at the post-mortem examination it was proved to be absent in nine cases.

From the above it will be seen that in class A fractured base is rare, in class B it is common, being present in two-thirds of all the case which died. This but bears out the recognised facts that the evil prognostic significance of fractured base depends upon the concomitant injury to the brain, and on this account it is of the same evil import in cases of compression as in cases unassociated with compression. The risks of septic infection of the clot, if on the same side as the fractured base, must not be lost sight of (*vide* cases 34 and 46).²

As regards the question of thickness of the skull, it can be settled with in a few words. The question that seemed to me worth considering was, whether in those cases belonging to class A where fracture is not commonly found, the thickness of the skull might account for this, and a blow which in the average thickness of a skull would fracture it, in these cases is sufficient to rupture vessels but not enough to fracture the bone owing to its inordinate thickness.

A reference to the cases shows that observations upon the bone are very seldom made, and even where they are the thickness of a bone is a relative measurement. Where the thickness is given, it would be important to know whether the bone is thicker at that spot than in a normal skull, for a skull might have an abnormally thin squamo-occipital and yet as regards the thickness of the bone, it might be greater than that of some parts of the lateral region (see Mr. Jacobson's paper on Middle Meningeal Hæmorrhage, with plates). Other points, such as the density of the cancellous tissue of the diploë, the proximity of sutures and the elasticity of the bone must necessarily enter into

² Possibly occurring in Guthrie's Case of Bilateral Subdural Hæmorrhage where one side suppurated.

the question. It is noticeable that under class A are classified those cases in which laceration and contusion of the brain are inappreciable, and hence we should imagine the violence to have been comparatively slight, and, as pointed out above, it certainly is much less than is found in cases coming under class B, and the probability is that in most cases at least it is not the conditions of the bone, so much as those of the blood-vessels, which are the important factors in producing extravasation without fractures. The cases coming under class A, in which thickness of the bone is mentioned, are Nos. 11 and 21, but the details are very meagre; on the other hand, that the thickness of the skull may be of some significance is shown by cases 47 and 59, belonging to class B. It is in reference to the latter case that Mr. Jonathan Hutchinson says, "It seemed probable in this case the great strength of the calvaria had permitted the reception of a very severe blow or fall, without fracture, and that in consequence blood-vessels had been ruptured at various places, and the lateral sinus given way." There is one lesson to be learnt from these cases, which is, that even if the bone be exposed and no signs of fracture detected, it does not follow that there is no extravasation beneath the bone.

4. THE CONDITION OF THE BRAIN.

As has been mentioned above, the division into classes A and B has been decided upon the condition of the brain. In class A there is every reason to believe from the clinical or post-mortem evidence that the brain had not been seriously damaged by the injury, and that if the compressing blood had been removed, recovery would have followed. In class B, on the other hand, in most cases the compression by the extravasated blood has been of secondary importance compared with the concomitant injury to the brain. Of the cases belonging to this class, which recovered, in four there was definite laceration found at the operation, but the patient survived this; in one (case 67) there is no mention of the condition of the brain found at operation, but the fact that some mental or motor impairment remained, points

to some cerebral lesion, and the last case (No. 64) which recovered, the patient was operated on for compound depressed fracture, and although no mention of laceration is made, I thought from the nature of the case it was best placed under class B.

The following is a brief summary of the condition of the brain in cases of class B:—

a. Marked laceration of the brain was present in fifteen cases. Of these fifteen cases, twelve are from the evidence found at the post-mortem examination, and three are from the observations made during the operation. In twelve of these fifteen cases the frontal or temporo-sphenoidal lobes (anterior extremities) were lacerated separately or together. In two cases the motor region was lacerated, in one opposite the anterior and middle fossa, and in the other it was found at operation beneath the blood clot. In one the position of the laceration is not stated.

In addition to these fifteen cases in which laceration was present, there were three others (*viz.*, cases Nos. 54, 69 and 70) in which some laceration was present.

(In case 54 there was a "lacerated hole one-third of an inch deep and one inch in diameter over the second and third temporo-sphenoidal convolutions traversed by a vein which was the probable source of hæmorrhage. In case 69 there was perhaps a slight cortical laceration of motor region beneath the clot." The patient recovered. In case 70, "Hole in posterior part of left superior temporal gyrus, into which a probe could be passed one and a half inches into gyrus at junction of middle and posterior part of convolution." This patient recovered but he suffered from albuminuria and granular casts, and I am inclined to look upon the case as one primarily of hæmorrhage into the brain which then broke through extra-cerebrally).

b. Contusion of the brain (local bruising) was present in seven cases. In four of these seven cases the frontal or temporo-sphenoidal lobes were the parts bruised. In three the position of the bruising is not mentioned, but from what the report says it is probably over the motor area in two of these.

c. Of the remaining eleven cases belonging to class B. In four, no post-mortem examination was made. In three of these extra-dural hæmorrhage was present in addition to the subdural. In three there was general concussion (in case 59 this was associated with pontine hæmorrhage). In two cases the patients recovered (*vide* cases 64 and 67 above), and in two cases which ended in death there is reason to believe that there was some injury to the brain from the presence of delirium.

Reference will again be made to these points in discussing prognosis. We have now to pass on to a consideration of the origin of the blood in these cases. I shall commence by giving a list of the possible sources and then proceed to a consideration of each individually. The possible sources are:—

a. The main arteries entering the skull, viz., the internal carotid arteries, and the vertebral arteries of either side. The basilar artery may be considered under this head.

b. The meningeal vessels.

c. The sinuses of the dura mater.

d. The vessels of the pia mater and the continuation of these which run across the subarachnoid space (the veins pass chiefly into the longitudinal sinus, the arteries are branches of the internal carotid and vertebral arteries but are considered separately here for convenience).

e. The cerebral vessels.

At the outset I may point out one difficulty in deciding upon the origin of the blood in cases coming under class A, the only group which presents difficulty, and that is that at operation in most cases, the clot is turned out, no bleeding point is found, and as the patient recovers no further investigation is possible; not only is this so, but in cases which die, in some cases a most careful post-mortem examination has failed to give any exact localisation of the source of the hæmorrhage. Mr. Henry Morris (*Lancet*, November 11th, 1882), drew attention to this, for he says, "If there be no fracture of the skull, no injury to the dura mater, no tear in the visceral arachnoid, no bruising or laceration of the brain, it is impossible to say whence the effused blood is derived."

a. With regard to the origin of the blood from the main arteries entering the skull. This is extremely rare, I have no record of the clot being due to this cause in any of the seventy-two collected cases, and nowhere can I find reported a case of rupture of the vertebral or basilar artery due to trauma; on the other hand, I have found two cases reported of rupture of the internal carotid artery, with subdural hæmorrhage, but, as was to be expected, both were fatal.

In the year 1876, James F., 17 years old, was admitted under Mr. Bryant. He had fallen from a wharf on to the deck of a ship, a distance of twenty feet. He had bruising of head and swelling of eyelids. He spoke a little after admission but soon became comatose. Pulse 102, gradually rose to 170. Respiration failed and pulse beat for two and a half a minutes after the last breath. The left side was thought to be paralysed. He lived three and a half hours. At the post-mortem examination (No. 68 for 1876) a fracture of the vertex extended into the base and passing across the left carotid foramen ruptured the artery, leading to extravasation of blood all through the arachnoid over the cerebellum. The left anterior lobe was torn through and the ventricle full of blood.

Another case is reported in the *Lancet*, October 24th, 1894, p. 912, "Case of Fracture of Base of the Skull, Rupture of the Internal Carotid Artery, Necropsy." Under the care of Dr. J. W. Stenhouse (Leith Hospital). Hæmorrhage was subdural over the right side of the cerebrum, and the whole of the anterior and middle fossa of the skull base. The patient lived almost four days after the accident. It occurred in a woman, aged 70, a chronic alcoholic. The right internal carotid artery was ruptured and a clot the size of a pea was found in the pons. Such cases are undoubtedly rare.³

b. The meningeal vessels. This practically resolves itself into an investigation on the position of the blood in cases of rupture

³ Mr. L. B. Rawlings, in his recent Hunterian Lectures on "Fractures of the Skull" (Lecture No. 2, abstracted in the *Lancet* of April 16th, 1904), reports two cases of rupture of the Internal Carotid Artery, within the Skull, both leading to immediate death, the blood escaping externally along the line of fracture.

of the middle meningeal artery within the skull. This is usually extradural, and in most cases is associated with a fissured fracture of the skull, which is the cause of the tear in the artery. The artery is torn in the groove on the bone whilst the dura mater remains intact. If, however, the dura mater is torn at the same time as the artery, the blood will be subdural rather than extradural, since it is far easier to separate the arachnoid from the dura mater than the dura mater from the bone. At the same time we should expect a small amount of blood to be between the dura mater and the bone since, as shown by the experiments of Sir Charles Bell, a small area of dura mater is separated from the bone at the site of the external blow. Cases No. 50 and 66 in Mr. Jacobson's papers were of this nature.

Of the cases upon which this paper is founded, No. 29 was also of this kind, and Mr. Mansell Moullin thought that, in the case he reported (No. 5 at the end), the source of the blood was the middle meningeal artery. That is to say, that of the seventy-two cases reported in this paper, one was definitely due to rupture of the middle meningeal artery, and one was probably due to this cause. I found one other case reported. This was in the King's College Hospital Reports, vol. ii. p. 135. The patient was admitted under Mr. Barrow on October 13th and died October 16th. The sternum, right clavicle, and six right upper ribs were fractured, and the second to the fifth on the left side. At the post-mortem the kidneys were granular and the heart atheromatous. His age was sixty-eight. It seems justifiable to conclude that although subdural hæmorrhage may be due to rupture of the middle meningeal artery, such an origin is rare.

c. The sinuses of the dura mater. Of the seventy-two cases collected, the source of the hæmorrhage was undoubtedly a sinus (lateral) in one (No. 20). In this case the blood filled the subdural space on both sides of the brain, and spread down to the vital centres. In one case the lateral sinus was ruptured and the brain also lacerated (No. 43) in one case (No. 59), the source of hæmorrhage may have been the lateral sinus. In considering case No. 20 it is interesting to note what Sir B. Brodie wrote in 1828. In a paper on "Pathological and Surgical Observations

relating to Injuries of the Brain," published in vol. xiv., p. 237, of the Pathological Society's Transactions, he says, "Wounds of the sinuses sometimes bleed profusely where there is a free opening in the bone made by accident or operation through which the blood can readily escape. But a very slight pressure is adequate to the suppression of this, as well as of other venous hæmorrhages, and I have never known an instance in which there was such a collection of blood as was capable of interfering with the functions of the brain, between the dura mater and the bone, or between the dura mater and the brain, in consequence of a wounded sinus." The common situation of blood due to rupture of a sinus of the dura mater is between the bone and dura mater. I refer the reader to a paper published in the Annals of Surgery for 1901, entitled, "Wounds of the venous Sinuses of the Brain," an analysis of seventy cases, by Henry R. Wharton, M.D. In this paper I found that subdural hæmorrhage was undoubtedly present in seven of these cases. In three of these the dura mater was torn clean through and in four it was only torn on its visceral side, *i.e.*, the inner layer of dura mater which limited the sinus was torn through, whilst the outer layer remained intact. On the other hand, there are only eight cases in which the blood from the sinus was definitely extradural. In "Revue de Chirurgie," September 10th, 1899, is a paper by MM. Gangolphe and Piery, on lesions of the lateral sinus based on eight cases, including one of their own. They consider that the clot is usually extradural, but frequently there coexists a hæmorrhage into the arachnoid cavity.⁴

d. The vessels of the pia mater and the continuations of these which run across the subarachnoid space. Of the eight cases out of the seventy-two cases reported, in which the source of

⁴ Mr. L. B. Rawlings, in his recent Hunterian Lectures on "Fractures of the Skull" (Lecture 2, abstracted in the Lancet of April 16th, 1904), comes to the conclusion that the blood extravasated from a sinus as the result of its rupture is "mainly intradural."

For Cases of Extradural Hæmorrhage due to Rupture of a Sinus," see vol. i. of the Pathological Society's Transactions, pp. 183 and 186, also vol. x., p. 167. Cases 76 and 77 in Mr. Jacobson's paper are also examples of extradural hæmorrhage due to rupture of a sinus.

the extravasated blood is given, in five it is said to come from a vein or artery of the pia mater, but the most powerful evidence in favour of this origin of the blood is that in those cases in class A where post-mortem examinations have been made, in the majority of cases no rupture of a meningeal artery or sinus has been discovered, laceration of the brain is entirely absent, and the pia mater itself is usually intact. In these cases I think it is right to conclude the source of the blood is a vessel in the subarachnoid space.

We are compelled, unfortunately, to fall back upon negative evidence and the problem as to whether the blood is usually arterial or venous in origin seems to me, in the absence of data to rely upon, to be beyond even venturing an opinion. The following, however, may be considered:—

(1) The condition of the clot. This is described as black, dark, or venous, if it is mentioned at all (eight cases out of thirty-six belonging to class A). This points to a venous origin, but the explanation may be the same as that put forward by Mr. Jacobson in his paper (see pp. 161 and 162, *Guy's Hospital Reports*, 3 s., vol. xxviii., 1885).

(2) If the rupture of the vessel be due to its stretching when a violent movement is given to the brain inside the skull, the arteries having more elastic tissue than the veins, would be less likely to give way as a result of the stretching.

(3) Against a venous origin is the long lucid interval which may be present. It is almost inconceivable that a vein can start to bleed three weeks after its original rupture, and whereas in the case of an artery a late hæmorrhage is capable of explanation on the supposition that an aneurysm has formed at the injured site perhaps associated with loss of support owing to destruction of the brain in the neighbourhood either from laceration or, in the case of contusion, from thrombosis of the vessels, no such explanation is possible in connection with late extravasation from a vein.

(4) Against a venous hæmorrhage also is the fact that to get the marked compression of the brain which is often present, we have to suppose that the extravasated blood forces the blood

out of the arteries of the corresponding hemisphere: in other words, the venous blood-pressure is capable of becoming greater than the arterial. This is against all known facts, and on the contrary, Cushing has shown (Mütter Lect. *vide supra*) that the blood-pressure is raised in order to overcome the compressing agent and "the centres are again nourished," and so on if the intracranial tension be again raised until the arterial pressure be forced two to three times above its normal level. This, however, as Dr. Cushing points out later, depends upon the integrity of the vascular control of the great splanchnic field, *i.e.*, to say that, in severe shock where this vascular control is lost, this compensatory rise in blood-pressure would not occur. But it is to be remembered that it is during "shock" that extravasation is usually deferred, and only as the patient rallies that the compressing blood flows out of the wounded vessel, *i.e.*, that nature brings into play the compensatory mechanism at the same time that the evil begins to act. On the other hand, it will be seen that Cushing's experiments point to the possible formation of a vicious circle if the blood be extravasated from an artery, for the pressure of the clot which leads to stoppage of the hæmorrhage is also compressing the brain. The vasomotor centre is brought into play and the arterial blood-pressure raised. This may dislodge any clot from the wounded artery, starting a fresh pouring out of blood which continues until once more the clot compresses, once more the vasomotor centre acts, and so on.

I venture the opinion, recognising, however, that the evidence I have put forward is purely negative, that the compressing blood is derived in most of the cases coming under class A, and those unassociated with laceration coming under class B, from the vessels of the pia mater and those running in the sub-arachnoid space. Further, from the experimental work of Dr. Cushing, I am inclined to look upon the blood as in most cases arterial or both venous and arterial in origin and rarely venous alone. In a small proportion of cases the blood is derived from the corresponding middle meningeal artery or one of the sinuses of the dura mater.

e. The cerebral vessels. By these I refer to the vessels supplying the grey and white matter of the hemispheres and in cases of laceration of the brain associated with subdural hæmorrhage, the most probable source of the blood is the ruptured arteries and veins.

THE SIGNS AND SYMPTOMS.

I propose to discuss these under the following headings, viz., Lucidity, Condition of the Pupils, Respiration and Pulse, the Temperature, Condition of the Limbs, Sickness, Delirium, Condition of the Urine, the Optic Discs. Although these signs and symptoms are thus grouped together it does not mean that they are all to be relied upon as leading to a diagnosis of subdural hæmorrhage, but merely that they are all worthy of consideration in dealing with head injuries, and an analysis of the presence or absence of such in the collected cases may at least tend to show whether any importance is to be attached to a particular sign or symptom.

"LUCIDITY" AND "LATENCY."

The lucid interval is so universally recognised that no further words are needed to describe it, but there is a condition to which I propose to give the words "latent interval" concerning which a few words of explanation are necessary. It is a well recognised fact that compression of the brain may occur without loss of consciousness but recognised by the appearance of paralysis, rigidity, or fits. In describing case 10 (q. v.), Dr. Kiliani mentions a "free interval" of twenty-one days which refers to the fact that the signs of compression of the brain did not occur for twenty-one days after the original accident. When I first gave the words "latent interval" to this interval which elapses between the original injury and the onset of symptoms of compression, unassociated with loss of consciousness, I had not seen Dr. Kiliani's case and had, in fact, habituated myself to the word latent as separate from lucid, hence my reasons for keeping to the word. That it is necessary that some such word should be used distinguishing between the onset of signs or symptoms unassociated with loss of consciousness and that of unconsciousness, becomes obvious when an

analysis of the reported cases is made. The one other question which arises is to decide where the latent interval ends. Does the onset of headache mean the termination of a latent interval? The safest position to take up, it seems to me, is to say that the latent interval ends when definite objective symptoms pointing to compression set in. Thus, with the onset of aphasia, paralysis, rigidity affecting one side and passing on to paralysis, fits, or a Hutchinson pupil, the latent interval terminates. In case No. 1 the latent interval of fourteen days terminated when "he developed a squint and soon became totally blind, began to lose power of speech and stammered a good deal." In case 23 the man lost consciousness suddenly a week after the accident and on recovering was "more or less speechless." In this case I have considered the latent interval as ending with the onset of the unconsciousness owing to the recovery of consciousness soon after and its persistence to operation.* It is probable in this case that the loss of consciousness was due either to sudden addition of a large amount of blood to a small primary clot, or to a sudden primary hæmorrhage. The early recovery of consciousness must be looked upon as due to some power of compensation on the part of the brain. In cases 6 and 25, and several others where the latent intervals ended with the onset of fits accompanied by loss of consciousness, but where between the fits the patients were quite sensible again, I have not looked upon the loss of consciousness with each fit as ending a lucid interval as opposed to a latent one. The matter may be briefly stated as follows:—The lucid interval ends when the patient merges into a state of unconsciousness which is only relieved by operation.

The following is an abstract of the conditions found in the seventy-two cases analysed:—

In class A.—

a. There was a "latent" or "lucid" interval present in thirty cases. Of these eleven died and nineteen recovered.

b. Of the remaining six cases in which no latent or lucid interval was present, three died and three recovered. (Of the three that died one was brought in dead, the injury was a blow

with the fist. The other two were almost identical injuries, viz., blows received whilst boxing).

a. Of the thirty cases in which a lucid or latent interval was present—

A latent interval was present in fourteen cases.

(In five of these the latent interval was terminated by the onset of fits, and during the fits the patient was unconscious. Consciousness was present between the fits.)

A lucid interval was present in thirteen cases.

(Of these thirteen cases, in one case, No. 4, the return to consciousness was brief and scarcely complete, whilst in case 12 the lucid interval was ended by death).

Of the remaining three cases, two had latent intervals which ended, but the lucid intervals still persisted for some days, finally ending in coma. In case 33, sudden hæmorrhage led to death on the ninth day without any previous warning.

As regards the length of time the intervals lasted, the following may be noted :—

Latent intervals :—

The longest is in case 10 where twenty-seven days elapsed.

The interval is under twenty-four hours in two cases.

The interval is over twenty-four hours and under seven days in eight cases.

The interval is over seven days and under fourteen days in one case.

The interval is fourteen days or more in three cases.

Lucid intervals :—

The longest is in case 21 where it is three weeks.

The interval is under twenty-four hours in seven cases.

The interval is twenty-four hours or more but under seven days in two cases.

The interval is seven days or more and under fourteen days in two cases.

The interval is fourteen days or over in two cases.

Of the other three cases—

In case 11 the latent interval ended on the second day after the accident, and the lucid interval persisted for another seven days ending on the ninth day.

In case 24 the latent interval ended on the twenty-fourth day, the lucid interval on the twenty-seventh day.

Case 33 is described above.

In class B.—

a. A lucid or latent interval was present in thirty cases. Of these twenty-four died and six recovered.

b. No lucid or latent interval was present in six cases. All these died.

Of group (*a*).

A definite lucid interval was present in nineteen cases.

(In case 60 the lucid interval was very brief. In case 62 it is to be noted that the patient lost much blood from a scalp wound, and the lucid interval lasted six days.

In case 68 the patient was twice bled immediately after the accident and remained conscious for three days when hæmorrhage suddenly came on and he died).

A definite latent interval was only present in one case (No. 72), but cases 51, 63 and 67 also had an indefinite latent interval.

Thus, in case 51 the patient was noisy and delirious after admission until the onset of fits. During the fits he was unconscious, but after the fits he could be made to answer questions.

In case 63 the patient had primary unconsciousness, from which he recovered. Two days later he became delirious and then aphasic. At the time that aphasia is first mentioned it is also said that the patient was almost comatose. Five days later fits set in. He still remained aphasic but conscious up to the operation, which was performed two days after the onset of fits. At the operation laceration of the brain was found beneath the clot. The patient recovered.

In case 67, there was a period of primary unconsciousness followed by delirium and excitement. No paralyses present. Morphia being given to induce sleep, twitching which ultimately became general convulsions, set in during sleep.

The remaining seven cases come under neither of the above headings and a brief abstract is given of each below.

In case 43, there was no definite lucid interval present, but on admission the patient could be roused. Early coma set in.

In case 48, injury December 30th. Delirious and wandering from December 21st to 31st. Coma January 1st. Death January 3rd.

In case 55, unconsciousness for two days, then brief return to consciousness followed by coma again.

In case 58, primary unconsciousness (21st). State of concussion from the 22nd to the 24th, then half comatose condition till death on the 24th of the following month.

In case, 64 primary unconsciousness. Lucid interval followed and the man was operated upon during the lucid interval for compound depressed fracture of the skull.

In case 69, primary unconsciousness for eight hours, then patient recovered consciousness, but was aphasic and remained so up to the time of operation. In this case it will be noticed that there was no latent interval but a lucid interval which only ended with the anæsthetic.

In case 72, irritable stupor gave way to violence, which was later succeeded by coma.

It will be noted that in the majority of these cases there was some approach to a lucid interval.

b. As regards the cases where no lucid interval was present, they may be considered as the most severe form of injury. They were all operated on save No. 61, but none of them derived much benefit from the operation. Fractured base was present in all those where a post-mortem examination was made, and in those in which no such examination was made there were signs during life pointing to fractured base. In two of the cases the blood was both extradural and subdural.

To sum up, it will be seen that the cases bear out the importance of a lucid interval in diagnosing traumatic extravasation of blood. The significance of this interval of lucidity has hitherto been emphasised in relation to extradural hæmorrhage but the above analysis shows that it has an equal importance when considering subdural hæmorrhage. As regards the presence of a latent interval, it may be asked why is it not present in cases of extradural hæmorrhage? I think the reply to this question is that the latent and lucid intervals end at practically the same time in this variety of blood extravasation. The latent interval is noticeable and important in cases of subdural hæmorrhage, in the first place, because it lasts such a long time; and, in the second place, because in a large number of cases the lucid interval persists up to the time of operation when anæsthesia is produced. This is so in all the cases classed under latent interval above, in not one of these was the lucid interval ended when the patients were prepared for operation, but in all the latent interval was ended by the onset of aphasia, fits, or paralysees, and thereby the diagnosis of compression of the brain made in most cases. On the other hand, in the analysis of the cases given above where the patient is described as having had a lucid interval it means that no aphasic, paralytic, convulsive or other objective symptoms preceded the onset of coma. An exception, however, to this may be granted in the case 29, where focal symptoms were present before consciousness was fully lost. In a study of the cases of subdural hæmorrhage as distinguished from extradural extravasation, two points are to be emphasised. They are probably more valuable from a diagnostic point of view than any others and may be looked upon as the most important which an analysis of the cases has elucidated; the first is the presence of a distinct latent interval as opposed to a lucid one, and the second is the long period of time which may elapse before the onset of any symptoms of compression of the brain in these cases, *i.e.*, the long interval of time which may elapse between the injury and the termination of the latent or lucid intervals. Before leaving this subject, I propose to discuss briefly two further points. Firstly, the reason for a latent period in one case and a lucid

period in another; and secondly, any explanations which can be given of the very long latent and lucid intervals which may occur. For a consideration of factors tending to cause bleeding and also the medico-legal importance of the lucid interval I refer the reader to Mr. Jacobson's paper (*vide supra*) pp. 260-264.

Experimental pathology shows that coma depends upon the anæmia of the brain resulting from the compression. This being so, we should expect the difference between those cases in which coma sets in without any objective symptoms preceding it and those in which objective symptoms associated with consciousness are present to depend upon the amount of the compressing force as a measure of the amount of blood forced from the cerebral vessels and leading to anæmia.

In the thirteen cases in which a latent interval was present, in seven the compressing blood, or blood and fluid, was small in amount. On the other hand, in four it was large. In one of these (case 10) it is reported as one and a quarter inches thick and practically covering the entire left hemisphere. Yet in this case the man never lost consciousness. It is possible that in the cases where the clot is extensive the brain may be atrophic and hence a considerable amount of clot be present without any marked compression. Failing this explanation, we must conclude that coma does not depend entirely upon the amount of the compressing agent, or if it does, in cases where the compressing agent is large, this has been gradually produced and allowed the brain to compensate. This power of compensation is rather suggested by case 10, referred to above. Here the accident was on May 5th. The patient had some prodromal symptoms on the 20th, on the 22nd he had "violent frontal headache," on the 26th "whilst walking from bed to table became dizzy and almost fell. This sensation passed away in a few minutes. Became weaker and unable to walk." On June 1st "slight hesitancy of speech" began, increasing till the time of admission, about June 7th. If the prodromal symptoms on May 20th be looked upon as the first onset of hæmorrhage and the maximum be looked upon as reached on June 7th, it means that blood was gradually poured out for eighteen days. There is no mention of any

appearance of the clot found at the operation, and suggesting that one part was older than another. The brain was very definitely compressed.

In cases with slow onset of hæmorrhage and gradual extravasation the explanation of absence of coma may be the same as in some cases of cerebral tumour, *i.e.*, that a corresponding atrophy of the brain occurs and allows the new growth (in this case blood) to take its place. (Allbutt's System of Medicine, vol. vii., p. 269, "Experimental Pathology of the Cerebral Circulation." By L. Hill.)

Turning to the question of why a latent or lucid interval should be present, many explanations have been given. A gradual hæmorrhage, as mentioned above, has been given as explanation. Prescott Hewett, writing in vol. xxviii. of the Medico-Chirurgical Society's Transactions (1845), p. 81, says, "Connected with these extravasations there is, however, one remarkable circumstance which having been already observed in several cases ought not, I think, to pass unnoticed. I allude to an intermission in the symptoms either of coma or even of paralysis. This intermission occurs under different circumstances; it may be dependent upon an interruption in the pouring out of the blood, during which the brain gets accustomed to the pressure and recovers its functions, until a further extravasation takes place. If carefully examined the extravasated blood will, in these cases, be found of different hues." He then proceeds to discuss the occurrence of intermissions also when the blood organizes and becomes enclosed to form a cyst.

Sir Benjamin Brodie, in describing the case reported at the end (No. 68), discusses the possibility of "secondary hæmorrhage" occurring in the cavity of the cranium and concludes that, although rare, this case is an example of it. He explains its infrequency by the "strictly antiphlogistic regimen usually pursued for a considerable time after the occurrence of the accident" (Transactions of Medico-Chirurgical Society, vol. xiv.). It is probable that reference is here made merely to what we should now call reactionary hæmorrhage, *i.e.*, bleeding coming on after a lapse of twenty-four hours. Since no sepsis was present

and no ligature used our present knowledge ascribes this "secondary hæmorrhage" to sudden forcible action of the heart, increased arterial tension, or pathological conditions of the walls of the vessels.

In discussing the case described by him in the *Journ. Amer. Medical Assoc.*, vol. viii. (reported as No. 22 at the end), Dr. Armstrong says, "pathologically the case is still obscure and the comprehensive term of intermeningeal hæmatoma was adopted as most closely covering the condition as evidenced. Agnew (*Surg.*, vol. i., p. 287) explains the condition as "a vascular paralysis so modifying the vital properties of the walls of the blood-vessels of the brain as to favour the free escape of their liquid contents." Considering the fluid character of the blood, and that, had the hæmorrhage been from a ruptured vein or artery, clot should have been present, this explanation is worthy of attention." A comparison with other cases, of which Mr. Butlin's is the best example, leads me to the opinion that, however ingenious the above idea may be, the more probable explanation is the effusion of serum round a small clot. The explanation of the late onset of symptoms may be explained by a recent collection of serum round an old clot which was not itself of sufficient size to cause compression symptoms. Even allowing the presence of such a "vascular paralysis" of which I do not know the counterpart in other injuries, save where inflammatory changes follow, its late onset seems difficult to explain.

Dr. Bremer, in considering the reason for the late onset of symptoms in the case which he and Dr. Carson published (No. 23 at the end) and in which he says the clot was old, suggests as a reason for the late onset of symptoms that the gradual hæmorrhage on the cortex had no effect at first but slight pressure. This irritates the vessels at the base and leads to afflux of blood through the lenticulo-striate artery. The cortex then becomes compressed between the clot on the surface and the increased flow through the basal ganglia.⁵

⁵ Compare with Cushing on raising of blood-pressure to overcome resistance of compressing agent referred to on p. 55.

In reporting the case which is No. 24 of the series, Mr. Hulke writes of "the hypothesis of hæmorrhage in this situation through bursting of a vessel consequent upon cerebral congestion as a late excessive reactionary effect."

It seems probable that in the majority of cases the late onset of symptoms means late onset of hæmorrhage, but it is a difficult matter at any time to say what the age of a collection of blood, within the skull, may be. It is to be noticed that of the ten cases belonging to class A, in which the latent or lucid interval is greater than seven days, in only three (Nos. 12, 22 and 24), is there reason to look upon the clot as old, and in two of these the compressing agent is "dark brown blood and fluid" in one (No. 22), and "brown flocculent fluid in another (No. 24). Reference to a paper entitled "Tumours containing fluid blood" by the late Mr. Marrant Baker, published in vol. i. of St. Bartholomew's Hospital Reports, may be referred to as throwing some light upon the possibility of so-called fluid blood being found long after its first extravasation and might possibly be brought forward to explain the presence of uncoagulated blood in some of these cases. He proves that in some cases fluid having every appearance of normal non-coagulated blood may be found in cysts of very old standing in which there can be no doubt that the extravasation is of old date. He then proceeds on experimental lines to show the reason for this, and on p. 218 writes, "the fluid is not simply uncoagulated blood but a mixture of serum (derived probably not only from extravasated blood but secreted also by surrounding parts) with blood cells, diffused colouring matter and disintegrated fibrine." In the cases under consideration, however, this explanation may be neglected for these reasons, firstly, that in the cases of long latent or lucid intervals clotted blood has usually been found, that in the cases of fluid blood being found it is extremely improbable that the above explanation is sufficient to account for it, since the changes leading to its formation, as pointed out in the above paper, in the series of experiments made, take many months; and lastly, that cases such as Nos. 22 and 24 are probably to be explained by a small clot and exudation of serum about

it, as pointed out by Mr. Butlin. For reference I have given a table showing the condition of the blood extravasation and the length of the latent or lucid interval in the ten cases in which this exceeded seven days.

TABLE OF REFERENCE

to Condition of Extravasated Blood found where the length of latent or lucid interval was over seven days.

No. of Case.	Condition of extravasated blood.	Length of latent or lucid interval.
1	Small blood-clot and some dark coloured blood which slowly welled up	14 days.
2	Dark semi-fluid clot was removed. Cerebral pulsations now recommenced causing more clot to collect	14 days.
3	Clot $1\frac{1}{2}$ inches thick covering practically the entire left hemisphere	27 days.
11	Black clotted blood exuded with each pulsation	2-9 days.
12	Blood fluid, or in the form of loose coagula. From reading the report it seems probable that there was an old part of the clot becoming organised and a more or less recent part in addition	11 days.
16	"Blood"	12 days.
21	"Clot" (microscopically showed "softening")	21 days.
22	A hypodermic needle drew forth dark blood "fluid" pressed out in larger quantity. (This "fluid" under the microscope consisted of "brown colored serum and colorless red blood corpuscles")	22 days.
23	Clot extending in all directions. On raising the dura mater semi-liquid blood forced itself through the superficial layer of the clot and reached 2-3 feet in height.	7 days.
24	Brown "flocculent fluid"	24-27 days.

It is of course possible that some cases may be explained by the rupture of aneurysmal dilatations of vessels which were damaged, but not opened at the time of injury. Very strongly against this is the fact that no post-mortem examinations

describe aneurysmal dilatations on vessels some days after injury either in the case of extravasation of the blood or laceration of the brain. It is useless to discuss the matter further, from whatever point of view the problem is approached it seems insoluble but it at least points to a moral and emphasizes the importance of careful post-mortem examination. In leaving this part of the subject it may also be pointed out that in some cases of early onset of coma also, we are compelled to conclude that factors are introduced which are outside our grasp, thus in the case reported by Mr. Mansell Moullin (No. 5) the man who was not stunned by the blow worked as a stevedore for four hours after the accident. To say that it took four hours for the shock to pass away and the heart and vessels to recover from any temporary depression seems absurd.

It seems probable on anatomical lines that a subdural hæmorrhage is more likely to slowly increase covering a long period of time, since whereas in the case of extradural hæmorrhage the blood has both to compress the brain and also separate the adhesions of the dura mater to the wall of the skull, a subdural hæmorrhage has practically only the former to do.

THE PUPILS.

The following is an analysis of the conditions found in the seventy-two cases reported :—

The pupils are not mentioned in	26 cases.
It is mentioned that they are equal and reacting in	10	"		
There was slight inequality of the pupils passing to				
a normal condition in	3 "
There was some abnormality of the pupils in	...			33 "
a. There was unilateral dilatation of the pupils in	7	"		
(But in only four of these was there a definite				
Hutchinson pupil.)				
b. There was bilateral dilatation in		9 "
(But of these in one case the pupils contracted				
later and in one the dilatation was only				
present during the fits (case 26).				
c. There was no case of definite unilateral				
contraction	0 "

- d. There was bilateral contraction in ... 3 cases.

(In one case it was pin-point and in this case unfortunately no post-mortem examination was made. In one case it is said that the one pupil responded to light and the other did not).

- e. The pupils are described as unequal in ... 10 "

- f. Of the remaining four cases:—

In case 66 the pupils were fixed and unequal.

In case 40 they were normal in size but non-reacting.

In case 53 a small local fracture of the middle fossa with hæmorrhage probably accounted for the various changes the one pupil underwent.

In case 45 the pupils varied. The right was greater than the left at first, then the left greater than the right, and finally the left was widely dilated and neither reacted.

The division into class A and class B shows the following:—

Of the twenty-six cases in which the pupils are not mentioned—

18 belong to class A ; and

8 belong to class B.

It is probable that in the majority of these cases the pupils were equal and reacting.

Of the ten cases in which the pupils are equal and reacting—

6 belong to class A ; and

4 belong to class B.

Of the three cases where practically no change is present—

2 belong to class A ; and

1 belongs to class B.

Of the thirty-three cases in which some pupillary change is present—

9 belong to class A ; and

24 belong to class B.

The typical Hutchinson pupil was only present in one of all the thirty-six cases coming under class A.

With regard to the mortality:—

Of the twenty-six cases in which the pupils are not mentioned—

16 recovered, and

10 died.

Of the ten cases in which there was no change—

5 recovered, and

5 died.

Of the three cases in which there was practically no change—

1 recovered, and

2 died.

Of the thirty-three cases in which an 'abnormality of pupils was present—

6 recovered, and

27 died.

Of the four cases of definite Hutchinson pupil all died.

Further examination of these four cases is as follows:—

In case 16, the pupil change occurred with onset of coma twelve days after admission. No operation was performed. The post-mortem examination states that the clot extended into the anterior fossa.

In case 52, both extra- and subdural hæmorrhage were present. Most of the extradural hæmorrhage had escaped through a fissure of the skull. After the operation it is stated that the right pupil was a shade larger than the left and that both reacted to light.

In case 60, an operation was performed but the dura mater was not opened. No extradural hæmorrhage was present. There is no mention of blood extending to the base of the brain.

In case 65, the pupil change came on twenty-four hours after admission. The patient was trephined, but died twelve hours later.

The following conclusions may be added:—

That of the cases in which the pupil is mentioned, abnormalities were present in 72 per cent. The real percentage, however, is

probably much lower, since the probability is that of the twenty-seven cases in which no mention is made of the pupils they were normal in the majority.

The abnormality of the pupils points to a bad prognosis, whilst a Hutchinson pupil is of particularly evil significance.

That the Hutchinson pupil is comparatively rare.

That the abnormality (inequality) of the pupils associated with simple concussion is to be distinguished from the abnormalities both as regards size and reaction to light occurring in cases of compression and associated with focal symptoms.

THE PULSE.

Class A.—

- a. There is no record of rate or character in ... 15 cases.
- b. Of the remaining twenty-one cases, there is a
pulse rate of under 65 in 6 "

(Of these six, the slowing of the pulse was associated with coma in two cases, viz., case 11, in which the pulse rate was 60, and case 17, in which it was 50. The former recovered after operation; the latter was not operated on and died.)

In the other four cases the patients were conscious at the time the pulse rate was recorded. It is necessary to go more carefully into these four cases:—

In case 23, there were signs of compression shown by paresis at the time that the pulse was slowed to 43. After operation, at which a large clot was evacuated, the pulse rose to 86.

In case 24, the patient was admitted with a pulse rate of 50 (very compressible) a fortnight after the injury, and at this time he had no focal symptoms. Signs of paralysis occurred on the fifth day in hospital, and, when on the seventh day he became comatose, the pulse rate was 70 (temperature 98°). Three to four ounces of brown flocculent fluid were removed from beneath the dura mater.

In case 29, the pulse rate of 64 on admission, soon after the injury, was indubitably due to shock, the patient at this time having no signs of compression. When these set in

nearly twenty hours later commencing with hemiparesis the pulse was 86, full and bounding.

Case 33 was anomalous. The hæmorrhage probably came on suddenly and was almost immediately fatal. The pulse rate was recorded on the day before his death, when no signs of compression were present. It was then 60.

This being so, we are, on the evidence supplied by our cases, compelled to acknowledge that in only two was there slowing of the pulse due to the compression and associated with coma, and in two others the pulse was slowed, the patient having focal symptoms but being quite conscious. In one of these last two, as the signs of compression increased and the patient passed into coma, the pulse rate increased from 50 to 70 per minute.

In two cases the pulse is described as slow and full. In the one it later ran up to 125 before the patient died, no operation being performed; in the other the patient recovered after operation, and, although the rate is not mentioned, the case may be perhaps added to the above as compression leading to slowing of the pulse.

The pulse rate was 100 or more in seven cases.

(Of these seven cases, six were comatose when the pulse rate was taken and of these, three were having fits, the pulse rates in these cases being respectively 160, 123 and 100.)

In one case the pulse was 70 on admission and became more and more rapid before death.

The pulse rate was between 65 and 100 in seven cases.

Class B.—

- a. There is no record of the rate or character in eight cases.
- b. Of the remaining twenty-eight cases:—

Frequency per minute of less than 65 in ten cases.

(Of these ten cases, in four the slowing of the pulse must be put down to shock. Thus:—

In case 40, the pulse was 58 and temperature 97° immediately after a severe injury.

In case 43, the pulse was 50 and the temperature 97° on admission.

In case 49, the pulse was 50 and feeble immediately after the accident, and there is every reason to believe that here also the slowing of the pulse rate was one of the signs of severe shock. The pulse later ran up to 180.

In the other six cases the slowing is probably due to the compression by blood clot or concomitant injury to the brain. It is necessary to consider these further:—

In case 48, the pulse was 58 when the patient became comatose, eleven days after the injury. The pulse rate reached 120 after the operation, at which two to three ounces of blood were removed. At the post-mortem examination contusion of the brain was found.

In case 52, extradural hæmorrhage was present in addition to subdural, the pulse rate was 51 (full), with coma. Post-mortem, laceration of the brain.

In case 53, the pulse was 40, laboured, and associated with Cheyne-Stokes' respiration half an hour after admission. Next day it was 60, rather small. This day the man was operated on and the day after his pulse rose to 96. Next day the pulse rate fell to 40, Cheyne-Stokes' respiration supervened and he died. Post-mortem, smashing of the anterior part of the right side of brain.

In case 56, the pulse rate was 54 on the fourth day after the accident. Post-mortem, bruising of brain and subdural hæmorrhage.

In case 63, the pulse was full and slow, 60 on admission, and though increasing in rate a little was still slow and full three days later. Recovery followed operation, at which clot was removed and laceration of brain found.

In case 71, the pulse was 60 and full one hour after admission. Clot was removed but patient died two hours afterwards, and at the post-mortem examination numerous small hæmorrhages throughout both hemispheres were found.

In one case the pulse is described as slow and regular on admission but later rose to 90 when paralysis was present.

Frequency per minute of 100 or more in six cases.

(All these patients died. In four of them bruising was found post-mortem without laceration. In one, laceration was present, whilst in the remaining case (54) although a small lacerated hole was found in the temporo-sphenoidal lobe beneath the clot it was thought that had the clot been removed the patient would have recovered.)

The frequency per minute between 65 and 100. Eleven cases.

(It is to be noted that in case 59, where the pulse was 72 and laboured, on admission the man had had a definite lucid interval, and at the post-mortem examination the brain was compressed by five ounces of blood subdurally and numerous ecchymoses were found in various parts of the brain.

Irregularity of the pulse :—

This is only mentioned once in class A, viz., in case 20. In this case from other signs and as shown at the post-mortem, blood had gravitated down around the medulla.

In class B, irregularity is mentioned three times (cases 45, 46 and 54). These all died. One of these cases (No. 54) it will be noted is the case mentioned above.

It will be remembered that in Mr. Symond's case the pulse was slowed with the onset of coma. This slowing in rate persisted until the time of operation. About a week after the operation irregularity of the pulse set in and lasted for some days.

As a summary of the above we may venture the following opinions :—

That whereas those cases coming under class A are to be looked upon as pure compression unassociated with concussion, contusion, or laceration, as far as this is possible after such violence, the fact that slowing of the pulse occurred in only four out of the thirty-six cases is against any significance being attached to the absence of slowing of the pulse in suspected compression⁶.

That if a pulse rate of between 65 and 100 be looked upon as a normal rate then in 37 per cent. of all the cases in which the pulse rate is mentioned (belonging to both A and B) the rate

⁶ In his paper, "One hundred Cases of Cerebral Tumour," published in the Guy's Hospital Reports for 1885-1886, Dr. Hale White calls attention to the infrequency of slowing of the pulse in cases of tumour of the brain.

is normal, but it was normal in a greater percentage of cases belonging to class B than belonging to class A. If, however, the pulse rate be looked upon as normal in those cases in class A, in which no mention of the rate is made, and it is probable that this is so, it would raise the percentage of cases belonging to class A in which a normal pulse rate is present to 60 per cent.

That of the cases coming under class B, in which contusion and laceration were present in only 16 per cent., is the pulse rate over 100 (*i.e.* six out of thirty-six).

That irregularity is of evil import, probably pointing to either severe laceration of the brain or gravitation of the blood to the region of the pons and medulla.

THE TEMPERATURE.

Class A.—

Of the fourteen cases which died :—

In five no record of the temperature was given.

In one the only mention is that the surface of the body was cool (17).

In one it is said that he “evidently had considerable pyrexia” (12).

In one case on the eighth day the temperature was 97°, on the ninth day the patient died, probably from a sudden large hæmorrhage (33).

In the six remaining cases there was some rise of temperature:—

In one it is said that with coma the temperature was slightly elevated.

In one the temperature was above normal from the first, and rose until at death it was 106·4°. At the post-mortem there was blood round the medulla (20).

In the other four there is a period of subnormal, normal, or slightly normal temperature followed by pronounced fever before death. Of these, in case 2 there was a period of sixteen days’ apyrexia; operation was then performed and the temperature afterwards rose to 101° between operation and death (no post-mortem made). In case 3 there was a period of eight days’ normal temperature preceding operation,

a period of twelve days' apyrexia followed, after which the temperature rose to 104° at death (compression by clot at post-mortem).

In case 5 the temperature was normal till the evening before death (period of ten days) when it went up to 109° , (post-mortem: compression by clot; trephining on the wrong side).

Of the twenty-two cases which recovered :—

In nine there is no record of the temperature.

In one (No. 14) the only mention is that two weeks after operation the temperature fell to normal.

In the other twelve the temperature is stated to have been over 98.4° at some period of the illness :—

In two of these it is practically normal, being 99° and 98.6° (on admission).

In one (No. 29) the temperature was 97.5° on admission (shock), but after the operation temperature rose to 110° F. during the night but immediately fell and gradually came down to normal.

Of the remaining nine cases there was fever prior to operation :—

In five of these cases the patient had convulsions (6, 18, 21, 23, 28) associated with the pyrexia and possibly accounting for it, although in case No. 18 the fever was also present before the onset of fits and apparently but little affected by their onset.

In one case there was some fever present but the temperature fell to normal later and the day after this fall fits set in.

In the three other cases (10, 22 and 34) no fits were present, in the last one (34) the fever, associated with delirium, preceded the second operation, at which a suppurating clot was found.

In four cases it is definitely stated that operation was followed by a normal temperature. These all recovered.

In four cases there was some fever following operation. These all recovered.

In four cases the temperature reached 104° or over during the course of the illness (6, 21, 27 and 29). These all recovered and are as follows :—

In case 6, the temperature on admission, six days after the injury, was 102° . This followed multiple fits for which he was admitted and which he continued to have up to time of operation. Four hours after operation temperature 105° and for four days varied from 101.4° to 104° . No fits after operation.

In case 21, the patient was admitted after a fit and had convulsions up to time of operation. The clot was removed at operation. It was said on the microscopical examination to be "breaking down." The temperature rose from 102° to 105° during stay in hospital preceding operation. Operation on twenty-second day. Four hours after operation temperature was 103° , on the twenty-fourth day it was 104° . On the twenty-sixth it was 99° .

In case 27, the temperature was 104° two days before onset of fits and normal the day preceding the onset of fits. No other record.

Case 29 has been referred to above.

Convulsions were present in fifteen cases :—

In eight of these there is no record of the temperature.

In three others no relationship between the fits and temperature is to be made out.

In four there was fever associated with the fits.

Class B.—

In fourteen cases there is no mention of the temperature. Of the twenty-two other cases :—

There was a temperature of over 104° F. in three cases. These all died.

In case 38, temperature rose to 107° before death.

In case 52, temperature rose to 104.6° before death.

And in case 66, temperature rose to 107° before death.

In ten cases temperatures from 99° to 104° are reported. Of these :—

In one it is between 99° and 100° (died).

In three it is between 100° and 102° (two lived, one died).

In four it is between 102° and 103° (all died).

And in two it is between 103° and 104° (one lived and one died).

In three fever was present but its degree not stated (45, 58 and 61).

No fever was present when the temperature was recorded in three cases :—

In two of these only one reference to the temperature is made, and in the other one there are two references. These references are, however, important, as will be seen from the cases.

In case 50, the accident was on the 10th. On the 15th the temperature was 98·6°. Death on the 17th. (Post-mortem, pulping of frontal and temporo-sphenoidal convolutions).

In case 54, accident on 5th. On 12th, temperature 98·4°. Died on 16th. (Post-mortem : No injury to general surface of brain but shallow local laceration of brain, one inch by one-third of an inch).

In case 56, accident 7th, admission 9th, died 12th. Temperature on 10th, 98·4, on 11th, right axilla, 95·8°, left axilla 97·8°. (Post-mortem : Superficial bruising of under surface of middle and anterior lobes).

With regard to the occurrence of pyrexia in association with laceration or contusion of the brain the following may be noted. In all the cases of lacerated brain where the temperature is recorded after admission there is some pyrexia, save in three, viz., 54, 55 and 61.

In case 61, there was a gradual rise, followed by a subsequent fall before death. Definite laceration was present at post-mortem examination.

In case 54, mentioned above, the laceration is extremely local and probably not to be classed with that usually described, whilst in case 55 it was superficial.

In the cases complicated with contusion the temperature is mentioned in five, and of these some pyrexia was present in four. In one case, 56, mentioned above, fever is absent as far as recorded temperatures go.

In all the three cases where the temperature was over 104° before death all had cerebral laceration at the post-mortem examination.

A reference to the various conditions found in the analysis of the temperature given above will be a sufficient reason for not drawing conclusions, and beyond merely epitomising the facts as follows, we shall discuss the matter no further.

That in what appears as the simplest varieties of compression a marked rise in temperature may occur and may even proceed to hyperpyrexia shortly before a fatal termination.⁷

That the presence of a mild degree of pyrexia is not of evil significance, since it occurred in 75 per cent. of the cases in which a report of the temperature was given in the cases of recovery belonging to class A.

That pyrexia may follow operation and yet the patient recover. (It seems probable that in these cases the fever persists from the first, but that should it set in on the third day it has the usual sinister meaning).

That in those cases where on admission or on their being seen soon after the injury the temperature is subnormal from shock but afterwards continuously rises to hyperpyrexia, the prognosis

⁷ Attention may be here called to Lecture III., on "Some points relating to Injuries to the Head," delivered by Mr. Battle before the Royal College of Surgeons of England, and reported in the *British Medical Journal*, vol. ii., p. 141, where the relationship between compression of the brain and pyrexia is considered. He refers to three cases quoted by Mr. Jacobson in his paper on Middle Meningeal Hemorrhage in which the temperature rose, and all had some laceration. He also refers to two or three where the temperature rose after operation. Passing on to Dr. Weisman's cases, he refers to twelve of these in which pyrexia was present: in six of these, one recovered, and no post-mortem examination was made on the other five; in the six others in which a post-mortem examination was made, contusion of the brain was found in four, one had a temperature over 102° before operation and died of pneumonia, and in the last the temperature rose to over 106° without any apparent contusion of the brain.

is very bad and may even be said to be hopeless, since severe laceration of the brain substance is present.

RESPIRATION.

Class A.—

There is no mention of the rate or character of the breathing in fifteen cases ; of the remaining twenty-one cases—

In seven it is mentioned that there was stertor with coma. In one of these (17) it is also described as interrupted, and the patient died two hours later “asphyxiated.”

In seven it is described as normal, or from the account there is every reason to believe that it is normal. In none of these cases was coma present.

In the remaining seven cases various conditions were present.

These are in cases 4, 6, 11, 18, 20, 21 and 31, and must be considered a little more fully :—

In case 4, the accident was on the 28th at 6.30 p.m. At 9 p.m. on the 29th there was Cheyne-Stokes' respiration, pulse feeble, 160, and the woman had been having fits for twenty-four hours. Her husband said that she suffered from epileptic fits and was a heavy drinker. At the post-mortem examination there was a little blood in the middle fossa by gravitation from a clot of two ounces over the left hemisphere. There was no blood round the pons or medulla nor was there blood intracerebrally. The kidneys were diseased, and the vessels at the base thickened and opaque but not aneurysmal. In this case the Cheyne-Stokes' character of the respiration may have been due to the renal disease. It may be noticed that Cushing, in his Mütter lecture, looked upon Cheyne-Stokes' respiration and the Traube Hering waves as due to the straining of the vaso-motor centre to overcome the conditions tending to produce bulbar anæmia. This almost certainly is the explanation in case 20 reported below, but I do not consider it as a probable explanation of this case.

In case 6, there was cyanosis with the fits, otherwise normal.

In case 11, loud and stertorous, with convulsions, otherwise normal.

In case 18, there is no mention save that the diaphragm worked spasmodically with the later fits.

In case 20, there was first stertor. Later a tendency to Cheyne-Stokes' respiration followed again by stertor. At the post-mortem examination blood was found to have gravitated around the medulla.

In case 21, the rate only is mentioned.

In case 31, the respirations ceased when the patient had a fit and when this was ended several deep breaths were taken.

Class B.—

There is no mention of rate or character of the breathing in six cases. Of the remaining thirty cases:—

In twenty stertor is mentioned as being associated with coma.

In one it is thirty to thirty-five, forcible, with coma.

In one case (60) it is definitely said that there was *no* stertor when coma was present. In this case the patient had been bled before Mr. Hutchinson saw him, and his face is described as pale and pulse rapid and feeble. The lucid interval was very short.

Cheyne-Stokes' respiration is mentioned as being present in two cases (Nos. 53 and 67).

In case 53, the patient first had stertor with coma, later there came Cheyne-Stokes' respiration. After operation, at which a quantity of blood was removed, the respiration was again stertorous. The Cheyne-Stokes' character recurred again before death. At the post-mortem examination there was a fracture of the vault and a small local fracture in the middle fossa at the left side. No hæmorrhage about medulla or pons. The anterior part of the brain on the right side was smashed. Organs healthy.

In case 67, the respiration was Cheyne-Stokes in character before operation. The skull was trephined, a large flat blood clot, found over the right motor area, removed, and recovery followed.

The other references to the respiration are unimportant.

CONDITIONS OF THE LIMBS,

Including the presence or absence of aphasia.

I have thought it best to arrange the signs present under the headings given below. The division into class A and class B is adhered to.

Class A.—

a. Aphasia was present in eight cases (1, 7, 10, 11, 18, 23, 28 and 30). In all these cases the aphasia was associated with some change in the condition of the limbs, paresis, paralysis or convulsions.

b. Paresis or paralysis unassociated with convulsions occurred in eleven cases. Of these:—

Consciousness was present in ... 6 (1, 3, 7, 10, 22, 23).

Five were hemiplegic and one monoplegic (arm).

Consciousness passing into coma was present in ... 2 (24 and 29).

(Both were cases of hemiplegia.)

Unconsciousness throughout was only present in ... 2 (5 and 34).

(Hemiplegia was present in one, no record is given in the other case.)

A condition of semi-coma with the obscure paralytic symptoms was present in the other case (32).

c. Convulsions unassociated with paresis or paralysis were present in nine cases:—

These were Jacksonian in type in... 2 (18 and 36).

Unconsciousness during the fits with consciousness in between the fits was present in 3 (26, 27, 28).

Convulsions, at first, Jacksonian in type but later associated with unconsciousness were present in ... 1 (19).

Complete unconsciousness was present both during and between the fits in... 3 (4, 8, 21).

d. Paralysis or paresis associated with convulsions was present in seven cases :—

The convulsions preceded the paresis or paralysis in 2 (4 and 31).

The convulsions followed the appearance of paresis or paralysis in 2 (11 and 30).
(In one of these the fits were Jacksonian in type.)

The paresis preceded the convulsions and the paralysis followed in 1 (14).

No statement as to the precedence of onset of convulsions, or paresis or paralysis is made in... .. 2 (6 and 25).

e. There is no mention of any paralysis, rigidity, or fits in five cases (2, 9, 13, 33 and 35).

f. There are four other cases unclassified, viz., Nos. 15, 16, 17 and 20.

In case 15, there were twitchings of the muscles of the face and left upper extremity when the patient was in coma just before operation. At the post-mortem examination a large coagulum was found compressing the brain. No mention is made of any laceration. Atheroma of the aorta and granular kidneys were present.

In case 16, there were very slight twitchings of both upper limbs when the patient was in coma on 26th. At the post-mortem examination blood was found compressing the left side of the brain.

In case 17, the limbs were powerless and flaccid from admission till death, there being no spasm at any time. At the autopsy, a quantity of soft blood-clot was found over the left middle cerebral lobe with very small ecchymoses beneath the pia mater on the same side.

In case 20, there was rigidity of all the limbs, with spasm. At the post-mortem examination blood was present over the whole brain and had gravitated down to the pons and medulla.

It will be noticed that all these four patients died.

Class B.—

a. Aphasia was present in two cases (63 and 69). Both recovered after operation. The one was associated with Jacksonian epilepsy, the other with right facial paralysis and paresis of the right upper limb.

Dysphasia was present in one case (64). This was a case of compound depressed fracture which came in with left-sided paralysis of the tongue, possibly the cause of the difficulty in speaking. The man recovered after operation.

In case 70, the patient suffered from inability to name objects, following convulsions, which occurred a week after the accident. He recovered after operation.

It will be noticed that the above cases include four out of the six cases of recovery in class B.

b. Paresis or paralysis unassociated with convulsions occurred in ten cases :—

Of these hemiplegia was present in six (45, 47, 55, 60, 62, 65).

Monoplegia in two (61 and 69).

Diplegia in one (both arms seem paralysed) (53).

And general paralysis in one (72).

In case 48, not included above, there was slight unilateral facial paralysis but no difference in tonicity of limbs.

c. Convulsions were present in ten cases :—

In one of these cases the type was Jacksonian and associated with aphasia (69).

In four there was some paresis or paralysis between the convulsions (46, 51, 54, 56).

In one case there was rigidity of the right arm between the fits (67).

In all save one (57) the convulsions, though usually starting in one definite part, spread to the whole of the body. In this one case they were unilateral.

d. Rigidity is mentioned as being present in five cases (38, 39, 44, 52 and 66) :—

In four of these the rigidity was general, and in one of these it was preceded by clonic movements and associated with double facial paralysis.

In the remaining case (44) the left arm is described as twitching, and the left leg as rigid.

e. General twitchings are mentioned in one case (40). In this case there was pronounced extradural and subdural hæmorrhage with severe laceration of the brain.

f. There is no record of the condition of the limbs in three cases (55, 59 and 68).

g. Four cases cannot be classified as above.

In two of these (42 and 43) there was no paralysis or rigidity before the onset of coma. In one (49) the limbs were flaccid on admission, but about two hours later power in the left arm was regained. Coma the whole time. In the remaining case there were clonic contractions at short intervals of the right arm and leg and sometimes of the left arm, with persistent tendency of the face to turn to the left side (case 71). Post-mortem, general concussion was found.

All these four cases died.

VOMITING.

It seemed to me that it would be worth while making an analysis of the cases in which vomiting was present and those cases in which it was absent, in order to see if any connection between vomiting and compression might be suggested. Unfortunately there is no reliable evidence, since in only five of the cases belonging to class A was vomiting noted as being present, whilst the negative evidence is not given. It is probable, however, that in the majority of the cases in which no reference to sickness was made, none was present. Of the five cases in which the patients vomited, one died, and in this case the patient was only sick when on the eve of an attack of delirium tremens. Again, in class B, reference is only made to the occurrence of vomiting in eleven of the thirty-six cases, and in four of these the vomited matter was pure or mixed blood and associated with fractured base. No importance, of course, can be attached to this small amount of evidence, but I am of the opinion that vomiting and this form of brain compression have but little relationship. Of the evidence forthcoming

above in most cases the sickness occurred before or immediately after arrival at the hospital and probably denoted recovery from the primary concussion. It is noticeable that in those cases with a long latent or lucid interval the patient did not suffer from vomiting during this interval.

DELIRIUM.^s

This symptom has a threefold importance. In the first place, it may lead to the onset of hæmorrhage owing to restlessness or violence of movements, or increase the extravasation of blood if this be already present. Secondly, it may be an important sign of coexistent brain lesion, and lastly, it may point to acute or chronic alcoholism (drunkenness on admission or delirium tremens later.)

The following is an abstract of the cases :—

Class A.—

No mention of delirium in eight cases.

No delirium present or every reason to believe that none was present in thirteen cases.

Of these, there was no lucid or latent interval in three, there was an alcoholic history in five; eight lived and five died.

Delirium was stated to be present in thirteen cases :—

Of these eight lived and five died. One of the five that died was a case of delirium tremens, the only one reported.

In addition the following cases (all of which recovered) may be specially mentioned :—

Case 14, the delirium followed washing out of the stomach, and was followed by the onset of paralysis.

Case 21, delirium was present in the intervals between the fits.

Case 25, it followed operation.

Case 27, mentions that three days after the accident he was feverish (104°) and delirious. This was two days before the onset of fits.

^s The presence of restlessness, constantly tossing about in bed, with total disregard for surroundings, at the same time muttering indistinctly or shouting loudly, violent movements, efforts to get out of bed, etc., are looked upon as reasons for considering the patients delirious.

Case 34, the delirium was present after the first operation. At the second operation a glassful of dark red fœtid matter was evacuated.

Of the two remaining cases, 11 and 13, in the former the patient was restless and frequently moaned before passing into coma, whilst the latter was brought in dead.

*Class B.*⁹—

a. Cases in which it cannot be said that delirium was present or absent, 10.

b. Cases in which no lucid interval was present, 6.

c. Cases in which there is no reason to believe that any delirium was present (64 and 69), 2.

Case 64 was a case of compound depressed fracture of the skull for which the patient was trephined immediately after admission.

Case 69, the patient was conscious up to the time of the operation after eight hours' primary unconsciousness.

Both of these patients recovered.

Case 70 probably belongs to this group also.

⁹ In looking through the cases coming under this class, it is rather difficult to decide how to arrange them in order that a fair idea may be obtained as to the presence or absence of delirium. The trouble is that in many cases, from what I have seen, delirium as shown by mere restlessness in bed, with incoherent talking or muttering, may be overlooked by those who have charge of the case owing to their leaving the patient to be washed and looked after by the nursing staff. By the time they again come to the patient in these cases it will be found in a large number that they will have become stupid, the early indication of passing into coma. Again, my own observations lead me to the conclusion that cases of compression associated with laceration may give no signs of delirium until the primary concussion has to a great extent passed off, and in many of these cases as the signs of concussion pass away the signs of compression set in. What I think is often found is that the patient is admitted unconscious; he starts to "come round," perhaps becomes a little restless, objects to examination, wants to lie still and almost immediately after this begins to pass into coma, the result of compression. When I first abstracted the cases coming under Class B, I found a large proportion in which there is no delirium mentioned, and no reason to believe that any was present, but a more careful examination showed me that in most of these cases the absence of reference to delirium was more probably to be explained by the absence of continuous observation and a definite active lucidity in between the primary unconsciousness and coma from compression. This contention will be borne out, I think, by reference to ten cases—37, 39, 42, 47, 52, 56, 59, 60, 66 and 71—and hence the heading (a) which refers to these cases.

d. Delirium was definitely present in 13 cases.

In six of these there is a history of alcoholism, either from friends of the patient, or the patient was admitted drunk. In two of these six cases the delirium was probably that of delirium tremens.

e. In the remaining four cases (45, 55, 65 and 68) a short period of restlessness, incoherent talking, or condition allied to delirium was present.

THE URINE.

The reason the presence in, or absence from, the urine of albumen was considered, was with the view of considering the possibility of traumatic subdural hæmorrhage leading to the presence of albuminuria without renal or cardiac disease or intracerebral hæmorrhage.

The condition of the urine is only mentioned nine times in the cases coming under Class A. In six of these albumen was absent, whilst in three it was present; these were cases 6, 16 and 20.

In case 6, there was a dense cloud of albumen present on admission, when the patient was having fits at intervals of one to five minutes, his temperature being 102° and pulse rate 100. There is no record of the condition of the urine following operation.

In case 16, the man had a large quantity of albumen in the urine when he passed into coma twelve days after admission. Nine days before the onset of coma there was no albuminuria. No operation was performed, and the patient died. The post-mortem record states that the kidneys were large and capsules adherent in places. Beyond swelling of the cortex, probably "from slight infiltration," there was no evidence of disease.

In case 21, the patient was admitted after a fit in the street, and on admission the urine was 1020 with a slight amount of albumen, no casts, no sugar. He had frequent fits up to the time of operation, and temperature on admission was 102°, reaching 105° before operation. He recovered.

The paucity of the information probably points to infrequency of albuminuria, and it is to be noticed that of the three cases in which albumen was present, two may possibly be explained

by the presence of convulsions, probably leading to elevation of blood-pressure and some increase of temperature, whilst in the third case, the albuminuria may have been the result of kidney disease.

Of the cases coming under Class B, the condition of the urine is mentioned in ten cases, and of these albuminuria was present in four. In one of these (case 39) there was traumatic intracerebral hæmorrhage unassociated with fracture of the skull. No disease of the kidneys or heart was found at the autopsy, and a lucid interval preceded the onset of coma. In the second case (67) fits occurred, and albumen was found in the urine. The patient was operated on, the clot removed, and recovery followed, but some motor and mental impairment remained.

In case 69, there was a faint trace of albumen found in a man aged 39; urine alkaline, with specific gravity of 1035. At operation, a slight cortical laceration was found. Recovery ensued.

In case 70, the man was the subject of some form of chronic nephritis, and it is interesting to notice that recovery followed operation.

These cases, again, neither prove nor disprove, but attention may be particularly called to the fact that subdural hæmorrhage may be associated with the presence of albuminuria, and although the one cannot, from our facts, be said to be due to the other, and although the occurrence of the two together is not beyond the possibility of being due to coincidence, yet the fact that the two have been associated together in the same person, and yet recovery follow the removal of the clot, must not be lost sight of when considering diagnosis or prognosis. It may further be said that if multiple fits have occurred, the two cases coming under Class A and quoted above, point to their being a possible cause of albuminuria.

OPTIC NEURITIS.

It will have been noticed in reading the report of Mr. Symond's case, given at the beginning of this paper, that optic neuritis was found to be present, but cleared up perfectly after operation. It seemed to me worth while to find out what references were made to the optic discs in the cases reported. An examination

of the cases coming under Class A shows that in two cases the discs were normal (7 and 10) in one other the "ophthalmic examination is negative" (17), whilst in case 22 there was double optic neuritis which was of a higher grade on the side of the clot. In this case the accident was at the end of February, the examination on the 28th April, and the man was conscious up to the time of operation (May 1st). These are the only references to the optic discs made in cases belonging to Class A. In Class B the optic discs are mentioned three times, viz., in cases 50, 52 and 70. In case 50 Mr. Lane found double optic neuritis on the sixth day after injury; the patient died next day. At the post-mortem examination there was found extra- and subdural hæmorrhage associated with severe laceration of the brain. In case 52 both discs were found "completely blurred over" three days after the accident. Here, also, in addition to the clot there was severe laceration. In the third case (No. 70) the "slight choking" of the right optic disc may have been due to the nephritis present. The importance of the possibility of optic neuritis occurring in cases of subdural hæmorrhage is, it seems to me, that as the late onset of symptoms of compression may lead to a diagnosis of intracranial suppuration or possibly tumour, it is of some importance to remember that optic neuritis does not settle the diagnosis in favour of pus being present. Careful examination of the optic discs in cases of compression by blood would probably show that changes in the discs are not at all uncommon in accidents associated with this complication. Its explanation probably lies in the venous stasis brought about by the compressing blood, as pointed out by Cushing (Mütter Lecture for 1902).¹⁰ It must not be lost sight of, however, that optic neuritis may be found associated with concussion or with fractured base when no hæmorrhage is present (see Lecture II. of Battle's Lectures before the Royal College of Surgeons on "Some points relating to Injuries to the Head").

¹⁰ Attention may also be called to Dr. Fleming's paper in the British Medical Journal, February 21st, 1903, vol. i., p. 409, "Retinal Hæmorrhages as a diagnostic feature in fracture of the base of the skull and in subarachnoid hæmorrhage."

DIAGNOSIS.

In discussing the differential diagnosis of traumatic subdural hæmorrhage, it is not my intention to inquire into the causes of coma, but to briefly refer to those conditions which have been seriously discussed when the diagnosis has proved difficult. These are extradural hæmorrhage, intracranial suppuration, uræmia, idiopathic epilepsy, cerebral hæmorrhage and meningeal apoplexy.

With regard to extradural hæmorrhage, the following words from Mr. Jacobson's paper on Middle Meningeal Hæmorrhage, (p. 295), written when considering the differential diagnosis between extradural hæmorrhage and hæmorrhage into the cavity of the arachnoid may be advisedly quoted—"While the diagnosis between these two extravasations must remain for the present very difficult, if not impossible, the treatment must be the same," etc. The writer then justly points out that the diagnosis is usually made when the crown of bone having been removed by the trephine, "the condition of the dura mater will very likely point to the presence of blood beneath it." This is all that is really necessary to say upon the differential diagnosis of these conditions. They have these points in common, that there is blood compressing the brain, that if relief to the compression is not given the patients will most probably die, and the relief in both cases is afforded by the operation of trephining. With the present day antiseptic methods there is no need to vary the prognosis as the dura mater is opened or not. It is probable that in the case of a latent or lucid interval of over a week the diagnosis will cease to lie between the position of the blood with regard to the dura mater, whether it be external or internal to it, but will more probably raise the question of whether the compressing agent is blood or pus. At the same time the two following cases found in the Surgical Reports of Guy's Hospital would point to the late onset of signs or symptoms in some cases of extradural hæmorrhage also.

G. C., 15½ years of age, admitted under Sir Henry Howse in 1888 (Report 220). His history was a kick on the right side of the head and shoulder on June 4th. He was admitted to a hospital from which he walked out on June 11th, but was

obliged to return next day. He left this hospital again on June 22nd and was admitted into Guy's Hospital the same day at 6 p.m., when he had double optic neuritis, retracted head, twitchings in the left arm, opisthotonos, pain in the head, photophobia, deafness of right ear and continuous crying. Mr. Howse trephined him in the afternoon of the 26th and a clot was removed from between the dura mater and the bone. The report does not mention the amount of the clot. The patient recovered after a long convalescence.

In 1878, there was admitted under Mr. Cooper Foster (Report No. 271) a man of 30. The accident was on December 2nd, and, save for a series of fits on the 6th and 7th, in which the convulsions were general, he had no more symptoms pointing to compression. No operation was performed and the man ultimately died December 18th. At the post-mortem examination a rather thick cake of black clot was found extradurally on the left side, with considerable bruising of the brain. The pulse was slow from admission on the 2nd until the 7th, but on the 13th it was 100.

With regard to the diagnosis between this form of subdural hæmorrhage and uræmia, the question will be specially raised in those cases where convulsions follow the injury after the lapse of a long lucid interval. What was said about the albuminuria possibly resulting from the fits must not be forgotten. The spread of the fits from some definite part of the body is in favour of compression but the points upon which the diagnosis will be made will be multiple and the pros and cons weighed carefully before any opinion can be finally given. Mr. Henry Morris's case (No. 16) was treated as though the coma was uræmic in origin and the case may be cited as an example of the extreme difficulty which may be found in arriving at a diagnosis in anomalous cases. Beyond the coma it may be said that there was no sign or symptom pointing to compression, save a Hutchinson's pupil. Attention may also be called to the fact that therapeutic treatment (pilocarpine, hot air bath, and calomel) led to marked improvement. It was when reporting this case that Mr. Morris wrote, "They give rise to no symptoms which

are pathognomonic or which even suggest with any definiteness their presence. The symptoms of coma and paralysis which they excite may be delayed for an indefinite period and are liable to intermissions dependent on recurrence of the hæmorrhage, or in old cases upon the more or less rapid absorption and effusion of the cyst fluid. Even when immediate symptoms of compression occur neither the nature of the accident, nor the character of the injury to the head affords any clue whatever to the exact locality of the blood."

Another example of difficulty in diagnosis is in the case reported by Raymond Johnson and Risien Russell (abstracted as case 6), and I cannot do better than repeat their own words when reporting the case. They wrote, "In the consideration of the nature of the case two possibilities may here be mentioned. The first, which was not, however, seriously entertained, was that the convulsions were uræmic in origin, for the urine contained a dense cloud of albumen. The second possibility which called for more careful consideration was that the case was one of idiopathic epilepsy brought out by a traumatism in an alcoholic subject with a strong predisposition to the disease, for we learned that the patient's father had died of epilepsy and that a brother was subject to epileptic fits. In this connection it may be noted that, although the fits almost invariably commenced on the left side, the first movement was commonly the turning of the head and eyes to that side, a mode of commencement that is too common in idiopathic epilepsy to be regarded as of much value in the diagnosis of a gross lesion of the brain.

"Many fits, however, began by twitching of the left side of the face, and others by movements of the left hand, and this fact, taken in association with the weakness of the left arm, which was observed between the fits, the exaggeration of the left knee-jerk, and the presence of the extensor response on the same side, was regarded as indicating the existence of a definite irritation of the right cerebral hemisphere. The evidence at our disposal did not seem to justify the diagnosis of a focal lesion, in that the fits did not constantly begin in the same region, whilst all the other

phenomena observed could be as well accounted for by a general disturbance of the right hemisphere as by a limited lesion.

"In the consideration of the nature of the lesion it may be remarked that, had the onset of the symptoms followed rapidly upon the injury, no hesitation would have been felt in regarding them as the result of an extravasation of blood over the right hemisphere caused by the blow on the opposite side of the head, the position of which was indicated by a small superficial scalp wound near the left parietal eminence. It was possible to explain the late onset of the convulsions by regarding them as the result of a simple non-inflammatory œdema spreading around a contusion or hæmorrhagic focus in the cerebral substance; but the chief difficulty in accepting this explanation consisted in the absence of any sensory or motor phenomena during the six days preceding the onset of symptoms. Notwithstanding, therefore, the absence of any satisfactory source of infection, we were forced to the conclusion that the lesion was probably inflammatory in nature—in fact, a meningitis."

In Mr. Symonds' case a diagnosis of probably extradural pus associated with infective osteo-myelitis of the bare bone seen at the bottom of the occipital wound was made, and with this idea the patient was trephined here at the first operation. Nancrede in discussing the diagnosis between compression by blood and pus, wrote:—"A differential diagnosis can, under the most favourable circumstances, be only probable and in most cases impossible." It is also to be noticed that Sir William Stokes puts a long latent period as one of the points in diagnosis of traumatic subdural abscess ("*Operative and Clinical Surgery*," p. 200), and passes on to say that the condition associated with pus production may be apyrexial. These statements and cases are the best means of showing that the differential diagnosis may prove extremely difficult in many cases. The correctness or incorrectness of the diagnosis between the presence of an intracranial collection of pus or blood is not such a serious matter as that between compression by blood and uræmia or idiopathic epilepsy, since, whether pus or blood be looked upon as leading to the compression, the only relief will be by trephining.

The diagnosis between traumatic subdural hæmorrhage and the so-called meningeal apoplexy and idiopathic cerebral hæmorrhage may also be noted as extremely difficult at times.

The following cases reported by Mr. J. Wood with the title of "Meningeal Apoplexy" in Vol. xiii. of the Transactions of the Pathological Society (p. 1) will show how such difficulties occur.

The man was found insensible (3 a.m. November 3rd), with no movements on the right side. On the left side the limbs moved on sprinkling the face with water. The mouth was not perceptibly drawn to either side. No reflex movements on tickling foot or leg of right side. Right pupil largely dilated; left, if anything, contracted. No movements of right eyelid, but friends said that he partly opened the left.

November 4th. Coma the same. Stertorous breathing, flapping of cheeks. Died 2.30 p.m.

The history was that he was an intemperate man, that he was intoxicated on the night previous to being first seen, but was able to walk. On arrival home, he threw himself on the bed and complained of headache. He finally went to sleep (or became insensible), not showing any signs of consciousness from that time until his death.

Post-mortem: No marks of violence. Large clot in the cavity of the arachnoid on the right side covering the middle and posterior lobes. Surface of brain compressed. Clot extended to the base in the middle fossa and as far as the optic nerve, also covering the tentorium. Membranes healthy save for a patch of bright crimson colour on the inner surface of the dura mater, opposite the position of the largest portion of the clot. Effusion of lymph in this situation and an appearance of ulceration. The brain itself afforded no appearance of disease save that the veins were full of blood. The age was not given.¹¹

¹¹ As an example of the difficulty in diagnosis between traumatic subdural hæmorrhage and idiopathic cerebral hæmorrhage, reference may be made to a case reported by Sir John Ericksen in his "Science and Art of Surgery," vol. i., 10th edition, pp. 767—768. In this case symptoms of compression set in a fortnight after injury. The man was treated antiphlogistically but died in three days. At the autopsy there was fracture of the base of the left side and subdural hæmorrhage on the right side.

It is almost impossible in many of the cases to decide with any certainty upon the diagnosis when some conflicting piece of evidence comes forward, but in leaving these brief notes upon the difficulties of diagnosis, after again emphasising the fact that most of these cases have been treated as extradural middle meningeal hæmorrhage in the past, and will probably be so diagnosed in many cases in the future, the following may be stated as the most valuable signs of the lesion being due to compression by blood. The first of these is the presence of a scalp wound or bruise, recent or remote. The importance of the presence of an old scalp wound may be emphasised by such a case as that reported as number 21 (abstract of cases at the end of the paper). Here the man suddenly fell in the street, was taken to a hospital, where he remained entirely unconscious without any history, save that given by a scar, which was judged to be from three to six weeks old. He was trephined over the situation of this probable evidence of a previous accident and removal of a large clot resulted in recovery. In these cases where the onset of symptoms is so late the examination of the scalp in cases showing compression symptoms is especially valuable. The second important point to rely upon is the presence of a lucid interval. The importance of this symptom needs no discussion, but I think that attention may be called to one point, and that is that the length of the lucid interval may have some prognostic importance, in that the longer the interval lasts the better the chance of recovery for the patient at operation. It seems to me that, broadly speaking, this condition is borne out by an examination of the cases. This is to be looked upon as very far from a dogmatic statement, and whilst I hold that it will, on the whole, probably hold true, I do not lose sight of such cases as No. 93, reported at the end, where sudden death from a large extravasation occurred on the ninth day. Lastly, of all the other signs and symptoms given it is probable that the most important are the presence of a fractured base and pupillary changes, especially a Hutchinson pupil, which, however, must be looked upon as rarely occurring. At the same time it must be recollected that in the cases belonging to class A, in

which the diagnosis proves most difficult, fracture of the base of the skull is rarely present and pupil changes are not common.

TREATMENT.

"Trephine and trephine early" was the dictum of Mr. Jacobson in reference to the treatment of middle meningeal hæmorrhage, and this is the summary of the treatment of subdural hæmorrhage. In the presence of symptoms it is the only treatment which affords relief, yet a number of cases having died without the performance of operation makes it worth while to consider the cases more carefully from this point of view, after which the conditions found at operation, and the principles of treatment dealing with the removal of the compressing blood, etc., will be considered.

Of the thirty-six cases coming under class A, no operation at all was performed in ten; in one (15) an incision was made down to the bone over the position of the injury, but as no fracture was found the trephine was not used; whilst in one case (5) the dura mater was exposed it did not pulsate, an incision was made into it, when the brain immediately bulged and began to pulsate, no clot, however, being found. The patient died, and at the subsequent post-mortem examination the clot was found on the opposite side. This case will be referred to again later. The ten cases in which no operation was performed were cases 4, 12, 13, 16, 17, 19, 20, 31, 32 and 33. The reasons for operation not being performed in these cases are briefly stated as follows:—Fits, thought to be epileptic, a history of epilepsy being obtained (4); absence of symptoms up to practically the time of death (12 and 33); death immediately following blows, the man being brought into the hospital dead (13); late onset of symptoms with no reliable signs, a diagnosis of uræmia being probably made (16); early death with no very reliable symptoms (17); a diagnosis of blood extending down to and compressing the vital centres and hence a belief in the uselessness of any operation (20), indefinite history of accident and great probability of idiopathic cerebral hæmorrhage (31 and 32).

Of the thirty-six cases coming under class B, no operation was performed in ten (39, 50, 54, 55, 56, 58, 59, 61, 62 and 68). In

the majority the explanation of withholding the trephine probably is that severe laceration or contusion was diagnosed.

The question of where to trephine will depend upon, first the situation of the blood as shown by the position of the paralysis, the site of onset of the fits, if such can be determined, or the side of a Hutchinson pupil should such be present. In the second place, the position of the injury to the scalp or cranial bones will have to be considered in many cases where definite localising symptoms are not present. The references to the occurrence of the hæmorrhage beneath the position of the scalp wound or fractured skull, or its presence at the corresponding point on the opposite side of the skull (*i.e.*, at position of *contrecoup*) are important; whilst the few facts relative to the condition of the skull bones may also be considered, and among these particularly the fact that in many of the cases belonging to class A no fracture of the skull has been present at all. As before stated, the probability is that in many cases the patients will be trephined with the diagnosis of extradural hæmorrhages, the crown of the bone will be removed, and no clot being found the possibility of the blood being subdural will have to be taken into consideration. What are the points upon which this is decided? They are two in number, (1) the non-pulsation of the membrane, and (2) its colour. I have made an analysis of the conditions present in the seventy-two cases collected with a view to the reliability of these signs. The following is a table of the results:—

Class A.—

a. No mention of either colour or pulsation in thirteen cases.

b. No mention of colour but no pulsation present, or the dura mater is described as bulging out on removal of the crown of bone in five cases.

c. The dura mater is described as “brownish blue,” “rather black,” “bluish,” “blood seems under,” “dark colour,” “greenish hue,” “dark cloudy” in thirteen cases.

(Of these thirteen cases there is absence of pulsation or presence of bulging of the dura mater in nine, in one the

fontanelle was bulging, whilst in one case where no operation was performed the dura mater was described *at the post-mortem examination* as "flaccid" (No. 12).

d. Of the remaining five cases—In case 5, mentioned above, the dura mater was exposed, did not bulge into the wound, was incised; the brain bulged and at once began to pulsate. At the post-mortem the clot was found on the opposite side.

In case 11, it is said the dura mater seemed normal, but a point of increased resistance was found in the lower third. After an incision here black clotted blood immediately exuded with each pulsation.

In case 24, the dura mater was healthy, free, but no pulsation. On incision brown flocculent fluid came away.

In case 25, the dura mater was healthy. It was opened and a clot found no bigger than a finger-nail. Serum around the clot produced compression.

In case 29, the blood lay within the torn dura mater. The middle meningeal artery was the source of hæmorrhage.

It will be noticed that pulsation of the dura mater is not stated to be present in a single case, and that it is definitely stated to be absent, or the dura mater bulging in fifteen out of the thirty-six cases.

Class B.—

There is no mention of the dura mater either as regards colouration or pulsation in twenty cases. (Of these twenty cases, in ten no operation at all was performed; in one the skull was trephined on the wrong side; in two of them "fluid" and "red serum" came out under pressure.)

Of the remaining sixteen cases—In twelve the dura mater is described as bulging, tense or non-pulsating.

In one case (44) extradural blood was removed in quantity and the dura mater was exposed, barely pulsating, tense and dark.¹²

¹² Note in this case the presence of a quantity of extradural clot. It is possible that the "barely pulsating" may have been a slight immediate response to the removal of this extradural clot.

In two cases "fluid was thought to be under the dura mater." No reasons are assigned for this belief.

In the remaining case (60) the patient was trephined with the diagnosis of right middle meningeal hæmorrhage. No blood being found extradurally, nothing more was done. At the autopsy one and a half ounces of blood was found extravasated into the arachnoid on the side on which the trephine had been employed.

Of the same sixteen cases—In only five is there any mention of the colour of the dura mater. In four of these it was "dark," "blackish," "black mass beneath," and "somewhat yellowish opaque."

In the other case it appeared white, and in this case there was a small clot with a quantity of clear fluid surrounding it and compressing the brain.

Case 35 (reported by Guthrie) is interesting reading when considering the intracranial tension produced by subdural hæmorrhage and its effect upon the pulsation of the dura mater. The man, a grenadier, after a lucid interval, fell into coma and was trepanned, but no extradural clot being found nothing further was done.

"Five or six hours after the operation he spoke and answered some questions, took some nourishment, but relapsed shortly afterwards into a similar state of stupefaction. On removing the first dressing the cause of evil was made manifest, the dura mater had risen up into the opening made by the trepan and was above the level of the bone, which had given some relief to the compressed parts and had probably been the cause of the temporary amelioration which had taken place." The dura mater was opened, blood evacuated, and the man recovered. It is after recounting this case that Mr. Guthrie goes on to say, "I consider this tense elevation and the absence of pulsation to be positive signs of there being a fluid beneath requiring an incision into the dura mater for its evacuation. It is a point, scarcely, if at all, noticed in English surgery, although much insisted upon in France." That the tenseness and non-pulsation of the dura mater are far more valuable than the colour will be emphasised

by the fact that if colour be relied upon, two conditions at least may lead to error, one being that the compressing agent is not always blood, but may be blood and serum, or serum alone, when there will be practically no discolouration notwithstanding the presence of pronounced compression; the other, that a thin layer of blood over the surface of the brain, associated with severe contusion yet incapable of compression, may cause discolouration, and this may also appear to be present when merely caused by the very distended veins on the surface of the brain pressed against the membrane. Of course, in the cases of subdural hæmorrhage the cause of the tense dura mater is the excessive intradural tension which will also be present in cases of tumour, abscess, œdema, or large intracerebral hæmorrhage.

Before leaving the subject of tenseness of the dura mater reference may be made to the line of treatment to be followed when the dura mater is exposed, opened, and yet no compressing blood or fluid of any kind can be found. In these cases the brain usually bulges through and begins pulsating. Of course it is possible the diagnosis is wrong and that a collection of pus may be present intracerebrally, but two possibilities must always be kept in view, the one being that the exact situation of the blood has not been found, and that another trephine hole may be necessary. Under these circumstances the relationship of the trephine opening to the brain and the motor phenomena present have rapidly to be considered, and the introduction of a curved director into the arachnoid cavity for the purpose of examining the neighbouring areas will probably prove satisfactory. The other possibility is that the paralysis and lesion may be on the same side. A sufficient number of such cases have now been reported to make it always worth while considering such a possibility. I will refer to four cases in which the paralysis and lesion were on the same side. Mr. Mansell Moullin's case (5) is one. Another was reported by Dr. Dawbarn (*Transactions of the New York Surgical Society*).

The patient had a blow on the right side of the head. During the following week he became more and more irritable. Then partial left hemiplegia and left facial paralysis developed. Two

discs of bone were removed from the site of injury and nothing found. The patient died two days later. At the autopsy a large blood clot was found over the left motor area, between the dura and the skull. Examination showed subsequently that it was one of those very rare cases where motor fibres had not crossed.

In the *Lancet* for May 26th, 1894, p. 1302, is reported a case by Mr. G. E. Turgnam, "Fracture of the Skull, paralysis on the same side as the lesion."

The case was one of depressed fracture, in a girl, aged 5.

The last case to which I shall refer is reported in the *Wein. Klin. Rundsch* (abstracted in the *Journal of the American Medical Association*, December 4th, 1897, p. 1188, "Subdural hæmatoma with uncrossed motor nerve tracts." "After a fall, dangerous symptoms developed, all indicating a subdural hæmatoma on the left side. Trephined on the left side without any result. As a fatal termination was imminent trephining was performed on the right side and a hæmatoma found. Recovery was prompt and complete, demonstrating the existence of uncrossed nerve tracts."

It seems worth while from the above recorded cases to suggest that when the clot cannot be found on the one side after a careful search, the opposite side of the skull should be trephined and a careful search be made for it there.

In reading through the reported cases it will be found that the compressing agent in some cases was not only blood, either as coagulum or fluid, but in addition some clearer fluid was present. In some cases this "fluid" was quite clear, surrounding a small clot, as in Mr. Butlin's case (25), in others it was tinted red by the blood present and is then described as "red serum," such as was present in Mr. Symonds' case. My attention was first called to the importance of deciding what the explanation of this "serous fluid" was by Mr. Symonds, who remarked upon it when it was removed at operation. For a long time, beyond Mr. Butlin's case and his explanation of it and the fact that such fluid was present also in cases, 22, 24, 26, and 30 belonging to class A, and cases 37, 40, 41, 42, 45 and 46 belonging to class B, I could find no reference to the probable source and nature of this fluid.

Mr. Butlin explained its origin in the case he reported as probably being derived as an effusion, serous in origin, the result of the irritation set up by the presence of the clot. In the *American Journal of the Medical Sciences*, vol. 116 (1898), however, I found a paper entitled, "Subarachnoid serous exudation productive of pressure symptoms after head injuries," by George L. Walton, M.D., in which the presence of a clear serous fluid without blood or injury to the brain is described as a cause of cerebral compression. His words are as follows: "It not unfrequently happens in these cases that trephining over the area indicated by the paralysis shows negative results beyond revealing a tensely bulging dura, incising of which is followed by a free flow or rather gush of clear fluid, the brain beneath presenting no laceration or other abnormality beyond perhaps œdema." Dr. Walton then cites two cases; in one, a boy, aged 16, pronounced focal symptoms came on immediately after an injury, in fact the writer says, "The case up to this time had appeared very suggestive of middle meningeal hæmorrhage." Improvement, however, steadily took place and the boy recovered perfectly without any surgical interference. His second case, occurring in a child three and a half years old, was similar. He then quotes another case in which operation was performed and this condition found. After giving diagnostic suggestions he draws a series of conclusions of which an abstract is as follows. That the condition can occur directly at the site of injury or contre coup, with or without œdema of the brain substance; that meningeal hæmorrhage may be simulated; that the condition is not compensatory but represents ineffectual efforts to relieve tension; that the lesion is self-limiting; and, finally, that paralysis following a blow on the head is not a reason for immediate operation.

In looking through the surgical reports of Guy's Hospital, I have found several cases reported where operation for compression has been performed, clear serous fluid escaped, and the patient recovered. The following is an example:—

Year 1878. Report 464, Mr. Davies-Colley.—Charles F., æt. 38, fell twenty to thirty feet. Picked up unconscious,

brought up to the hospital and admitted October 6th. Remained unconscious until the 9th, when he developed fits on the right side. Trephined and a subdural collection of cerebro-spinal fluid found on the left side. No injury to the brain. The dura mater was not sewn up nor the bone replaced. Flap sewn up. From 11th to 14th had fits. On the 15th consciousness returned. (Urine 1020, no albumen.) Paralysis which had been present on the right side is said to have passed off on the 16th, but a little paralysis of the left side of the face remains. On October 20th the stitches were taken out, the wound healing by first intention, and the man went out soon after.

The next case is one of recovery without operation:—

Year 1881, report 216, Mr. Bryant.—This is the report of a girl, Eliz. C., 12 years old, who fell backwards down four steps on to the back of her head. She lay for a few seconds then got up, walked upstairs, and again became unconscious. She was admitted on June 20th, and was sick three times within twelve hours of coming in. She recovered perfectly without operation and was discharged on July 3rd.

This last case may be compared with one given by Guthrie.

P. T., 24, was struck on March 16th, 1837, by a musket-ball, on the frontal bone, which it fractured but did not penetrate. Walked into the ward from the field of battle, some two miles distant, and conversed intelligibly for some time to his comrades. Much hæmorrhage had taken place. Towards evening he became comatose; pulse slow, weak; surface pale and cold. Mercury, jalap and some pulv. ipecac. were given at 9 p.m. Noted that a small arterial branch had bled very freely, by which the symptoms have been relieved. On the 22nd, the pulse was 52, sluggish. He made a perfect recovery.

In this case the loss of blood probably had much to do with the recovery, since all the available fluid was, in all probability, immediately taken up by the vessels. This idea receives support from the case reported by Le Drans, and quoted by Guthrie with the above case. (Guthrie's "Injuries of the Head," p. 57.)

A case in which a diagnosis of ruptured middle meningeal artery was made was reported by Mr. Legg in the *Clinical Journal*, December 30th, 1903, p. 176, "Head Injuries."

In the *New York Medical Journal*, April 7th, 1894, p. 434, is a case reported of "Brain Cyst, the result of injury, causing aphasia, hemiplegia, etc. Evacuation; complete recovery," by C. H. Mayo, M.D.

O. S., female, 11 years, thrown from a waggon, June 30th, 1893. Picked up unconscious, bleeding freely from the nose, and while being carried into the house had a severe vomiting spell. There was a small scalp-wound and extensive bruising. She remained unconscious for six weeks; afterwards she began to notice things with her eyes. She had right hemiplegia, and on the fifth day after admission she had twenty-five convulsions, and these were continued for a week, becoming less frequent each day. On August 18th she was trephined over the left fissure of Rolando; four ounces of clear fluid was removed subdurally and the left Rolandic area was found to have been depressed one inch from the skull.

This case would seem to suggest that the self-limiting power of the effusion is not absolute.

A case of extradural clot in a thin layer with clear fluid subdurally is reported by Mr. A. E. Maylard. The case was brought before the Glasgow Medico-Chirurgical Society on November the 1st, 1895, and is reported in the *Glasgow Medical Journal*, February, 1896. It occurred in a girl of nine years old.

The effusion may occur without trauma. Two such cases are reported. One in the *Lancet*, September 22nd, 1900, "Trephining and Drainage in an apparently moribund case of Status Epilepticus: Recovery." (Under the care of Dr. W. Alexander, notes of the case by Dr. W. T. D. Allen, Sen. Resd. Med. Off.)

This was a woman, æt. 36, admitted July 4th, 1900, in a semi-conscious state, suffering from very severe epileptiform convulsions. From the previous history the patient appeared to have been in good health until the previous February, except for occasional attacks of articular rheumatism. She had been very much addicted to drink, and in February had been taken into

custody for this cause, and it is stated that the policeman then struck her on the head with his bâton. The head became swollen and she was treated in the prison hospital. After two weeks in prison she was removed to the workhouse, where she had an epileptiform fit which lasted fifteen minutes. The convulsions involved the limbs on both sides of the body and the patient was in a rather stupid condition for a day and a half after the fit; she made a gradual recovery in ten days. She had never had a fit of any kind previous to this time. During the next four months she was in fair health, save for a pain in her head, which was at times very severe. On July 1st, convulsions began again and continued at very short intervals for four days until she was taken to the workhouse hospital. On admission she was semi-conscious; she had over forty fits in twenty hours. These began with twitchings of the muscles of the lower part of the face on the right side, which then spread to the right hand, arm, and leg. Corneal reflex was absent and the eyes directed to the right. Each fit lasted three to four minutes. On July 5th the patient appeared moribund and was trephined by Dr. Alexander over the upper motor area on the left side. The dura mater bulged into the opening on removal of the bone and was opened. The cerebral veins were much enlarged and lay bathed in a layer of fluid a quarter of an inch thick. The fluid was held in the meshes of the arachnoid and pia mater, and when the meshes were pricked the fluid ran away in a clear sparkling stream; a gauze drain was used. After the operation, the patient had several fits and these continued for three days but gradually diminished in frequency and severity. Since then there has been no return of the convulsions or headache.

Of course, in this case it is possible that the whole trouble dated from the injury and that the symptoms varied as the fluid "ebbed or flowed." The other case is reported in the *B. M. J.*, October 17th, 1896, vol. ii., p. 114. It is the first of "Two cases of Jacksonian epilepsy treated by operation," by Albert E. Morison.

Subdural collection of straw-coloured fluid, but without any history of accident at all: Operation: Recovery.

From the above cases it would seem that in cases of subdural hæmorrhage the compressing force of the blood extravasated may be added to by the exudation of a serous fluid probably derived from the lymphatics. The irritation of the brain by blood-clot seems to be sufficient reason for this fluid being extravasated in some cases, but that other factors are introduced is certain from the cases quoted above. The date of the extravasation also seems variable; thus, in cases 22 and 24 there was a latent interval of twenty-two and twenty-four days respectively, and at operation brown serum was removed in each case. In these cases it seems probable that there was a small clot, much as in Mr. Butlin's case, but for some reason which for the present seems inexplicable, the serous exudation did not occur until the clot was undergoing degenerative changes, as shown by the brown colour. In case 24 the latent interval lasted up to the twenty-fourth day, but the lucid interval up to the twenty-seventh day, when the patient lapsed into coma. This is the only case coming under class A where coma supervened as a result of such exudation. In the other three cases—Nos. 25, 26 and 30—there were latent intervals respectively of four, six and two days.

There is very little more to be said upon the compressing blood. In the cases of marked hæmatoma it will lie in the cavity of the arachnoid,¹³ and will be exposed immediately the dura mater is incised. The probable reason for this is that as a result of the injury the delicate arachnoid is torn at the site of injury and extravasation, and even were this not so it seems probable that the force of the extravasated blood would cause it to break through into the loose cavity of the arachnoid (which, though only a potential space is capable of becoming a very large actual one) rather than constantly breaking down the

¹³ Sir Prescott Hewett speaks of extravasations of blood into the cavity of the arachnoid. Mr. Henry Morris reported his case as hæmorrhage into the cavity of the arachnoid. Mr. Jacobson, in his paper referred to above, differentiates between extradural middle meningeal hæmorrhage and hæmorrhage into the arachnoid cavity. The cavity of the arachnoid is thus generally recognised as the situation of the blood.

meshes of the subarachnoid space. The following is an analysis of the amount of the compressing blood or blood serum :—

Class A.—

The amount present is mentioned in ten cases :—

The greatest amount present was four ounces (case 12).

The least amount present was half an ounce (case 18).

The average amount present in nine cases was two and one-third ounces approximately.

In the tenth case twenty-five grammes were present.

The thickness of the clot is mentioned in three cases (10, 5 and 19), where it was respectively one and a quarter inch, three-quarters of an inch and one-sixth of an inch thick. In this last case the man had a definite lucid interval and focal symptoms. He was not trephined only because of the disadvantages of his position.

Class B.—

The amount is only mentioned seven times.

The greatest amount present was five ounces (case 59).

The least amount present was one ounce (case 14).

The thickness was mentioned on three occasions, when two and a half inches, one inch and half an inch were given.

The case in which the clot was two and a half inches thick had a lucid interval of at least twelve hours (case 65).

There is a case, however, reported by Sir B. Brodie in a paper to the Medico-Chirurgical Society (Transactions, pp. 347–348), in which the compressing blood measured eight ounces. The case is not classed in the seventy-two reported cases owing to the want of detail. The woman had a lucid interval of one hour. No operation was performed, the condition being found post-mortem.

One other point requires mention, which is the possible presence of two strata of blood, one of which may be removed and the other left, giving some relief which may only prove temporary. The best example of this is the case reported by Mr. Edmund Owen (case 18). Here at the first operation a large clot was most thoroughly removed from the cavity of the arachnoid. After the operation a quiet night supervened but

further progress was stayed by the onset of severe convulsions which led to the wound being reopened two days after the first operation; the arachnoid membrane was incised and some clot removed from the subarachnoid space. Gradual complete recovery followed.

In case 64 also there were two layers of clot.

The method of removing the clot will vary with the operation and the conditions present. Various methods have been employed by different operators. In case 1 a blunt spoon was used; in case 10 a finger and spoon; in case 27 the little finger and the scoop end of a director.

There is an interesting case reported in the Journal of the American Medical Association, vol. xxiii., p. 952, by Dr. J. B. Hamilton, in which the method of removing the blood showed great ingenuity.

Sister A., æt. 20, strong and healthy. Struck on the head just posterior to the right Rolandic fissure by a staple or hook, on June 18th. There was great pain, but no wound. Severe headache followed which never disappeared. Vision in the right eye gradually failed and she became totally blind in that eye. Deafness in the right ear began almost immediately after the accident and continued. There was no motor paralysis, no loss of sense of smell or taste. Vision and hearing apparently normal on the left side. Trephining November 15th. The dura mater was healthy; it was incised and a silver wire loop passed downwards and forwards towards the optic commissure. On withdrawal, a firm round coagulum about 3 cms. in length was found attached to the wire. The dura mater was sutured and the bone replaced, the wound being closed. Complete recovery followed.

Irrigation was stated to be employed in five cases. These were 18, 28, 42, 47, 69. Various fluids have been used, including boracic lotion, lysol, carbolised water and salt solution.

In the cases belonging to class A, it seems that the hæmorrhage is not difficult to stop, there seldom being any recurrence of the bleeding when the clot is removed, but in the cases belonging to class B severe and even dangerous recurrence of the bleeding may take place. This was so in cases 43, 44, 46, 47, 64 and 66.

In case 43, the blood coming from lower down in the subdural space seems to have been impossible to arrest, even by packing with gauze. The dressings were repeatedly soaked through with blood, and so great was the loss that the patient was infused just before death.¹⁴

In most of the cases of severe hæmorrhage a plug of gauze seems to have controlled it. In cases where severe persistent bleeding comes from low down its origin is probably from the lateral sinus. In case 71, a bleeding cerebral artery was tied and in case 40 a bleeding cerebral vein.

Lastly, the advisability of drainage will have to be considered. An analysis of the seventy-two cases is as follows:—

Class A.—

In the first place, it is to be noticed that of the fourteen cases which died, eleven had no operation at all; in one the operation was performed on the side opposite to the collection of blood and had no bearing on the result; of the two remaining cases, in one drainage was employed and in one the dura mater was sutured and the bone replaced.

Of the twenty-two cases which recovered:—

Drainage was definitely employed in ten.

A tube was used in three.

Gauze was used in two.

Horsehair was used in two.

The nature of the drain is not mentioned in 2; whilst in the remaining case the space was drained with a strip of gutta serena, the bone being replaced.

No drainage at all was used in one case (No. 1). In No. 6 also no drainage was probably used, the divided dura turned into position, the scalp incision sutured, the bone not replaced.

Two cases came in in the pre-antiseptic days and in these the dura mater was only punctured.

In case 18, some form of drainage was probably employed down to the dura mater, which was sutured.

In case 26, the dura mater was stitched, but the bone not replaced “because of bulging.”

¹⁴ Unfortunately no post-mortem examination was made.

There is no reference to the method of suturing or dressing in six cases.

It may be noticed that in case 28 "the wound was lightly packed with gauze and allowed to granulate." It might be thought that by this method there would be a risk of anchoring the surface of the brain to the bone round the trephine opening, it is therefore interesting to note that he made a perfect recovery, and three months after his discharge was working in a colliery where he had been for some weeks, and was doing perfectly well.¹⁵

Class B.—

Of the six cases which recovered, in five drainage was used, and in the other there is no mention as to whether it was used or not.

Of the other thirty cases—

There was no operation in	10
The dura mater was not opened after trephining in	...				1
There is no mention whether drainage was or was not used in	6
Drainage was definitely employed in	10
No drainage was employed in	3

These last three patients were Nos. 46, 47 and 66.

In case 46, the dura mater was stitched, the bone replaced, and the flap sutured in position. This was on January 25th. On the 10th day of February grave symptoms set in, the patient having done very well for the previous sixteen days. The wound was opened up and a cerebral abscess found. Death followed. There was no autopsy. A fracture of the base was undoubtedly present.

In case 47, the wound in the dura mater was closed, the bone being replaced in pieces, and the skin sewn up with a gauze drain. Death occurred three days later, and at the post-mortem examination a re-accumulation of blood was found.

In case 66, "as the bleeding came from some inaccessible source, the wound was sewn up and the man sent to bed. At

¹⁵ The fact that only three months had elapsed makes the value of this statement practically negative from a prognostic point of view.

the post-mortem examination, the subarachnoid space over the hemispheres was practically filled with blood."

It will thus be noted that of the two cases where it was considered satisfactory to do without drainage (viz., 46 and 47), in the one, had it not been for the accidental complication, there is every reason to believe that a satisfactory result would have followed, whilst in the other there can be little doubt that lack of drainage at least hastened a fatal result.

It would seem safe to close the wound entirely when all clot has been removed, no recurrence of bleeding has taken place, and expansion of the brain has followed, but no rules can be finally laid down, and every case will probably be treated from the special conditions found.

PROGNOSIS.

The importance of compression by blood seems to depend upon three points—the first, the rapidity with which the blood is poured out; the second, the amount of blood so poured out; and the third its position. The importance of the rapidity with which the blood is poured out is shown by cases such as Nos. 17 and 38, where the patient dies almost at once. Such cases may show a much smaller amount of blood extravasated than in many which recover after operation, but the rapid extravasation would appear to lead to sudden paralysis of the vital centres in the medulla, probably from the sudden transmission of the compressing force. That the amount of blood poured out is another important factor is again undoubted, and this acts by the transmission of the pressure to the vasomotor and respiratory centres in the medulla, whilst the importance of the position is that the nearer the clot is to the base the greater its effect upon these vital centres. Sir B. Brodie, with reference to the importance of the position of the clot, said, "There is reason to believe that pressure is on the whole more dangerous when it affects the lower part of the brain than when it affects the upper part."

The division of the seventy-two cases under consideration into classes A and B is a prognostic one. The former are pure

compression, and being such, the relief of the compression should mean recovery; the latter are associated with severe cerebral injuries, and, although the compressing blood may be removed, in most cases early death will usually follow, owing to the severe concomitant injury to the brain. In class A, of the thirty-six cases recorded, twenty-two recovered and fourteen died. The reasons for death occurring in these cases has been set forward briefly when considering treatment. It will be noticed that a bad prognosis would have been given in the majority. In class B, numbering also thirty-six cases, only six recovered. In these cases the laceration or contusion of the brain most probably accounted for the fatal result. Of the total seventy-two cases collected, therefore, twenty-eight recovered and forty-four died, *i.e.*, thirty-nine per cent. of recoveries. This compares well with the eight recoveries out of the seventy cases reported by Mr. Jacobson of extradural hæmorrhage from rupture of the middle meningeal artery, *i.e.*, 11·5 per cent. of recoveries.

It must be mentioned, however, that Mr. Jacobson goes on to say that five other recoveries are reported. But no statistics will prove of any real avail in giving a prognosis in cases of compression by blood clot in individual cases. What has to be considered on such occasions are the signs and symptoms of the case under consideration, and it is possible that no sign or symptom is so valuable in leading to a good prognosis as a definite active lucid interval, during which the patient has appeared comparatively well and acted as an intelligent individual, whereas no prospect will appear more hopeless than that where no lucid interval at all has been present, or if it has been present, has been brief and associated with a nervous irritability and irresponsibility, which bodes ill, pointing as it does to severe brain injury.

In people advancing in age hæmorrhage into the brain substance may occur in association with the subdural extravasation. Dr. Goodhart mentions such a case in his paper (case 32) on "Meningeal Hæmorrhage," published in vol. xxi. of the *Guy's Hospital Reports*.

A man, æt. 68, slipped on a step and fell a short distance, striking the left temple. Short lucid interval followed. He had slow breathing and slow pulse. Pupils could not be observed. At the autopsy, there was two ounces of blood in the left subdural space, and hæmorrhage into the corpus stratum and septum lucidum on the opposite side.

A case of subdural hæmorrhage, associated with pontine hæmorrhage, was admitted under Mr. Durham in May, 1891 (Report 341).

Mary A., æt. 51.—She had brought up a large quantity of blood six months before. She drank freely. She was found at the bottom of fourteen steps perfectly unconscious, and with stertorous breathing. She remained the same all night, save for some slight bleeding from the nose and mouth, and was sent up and admitted next morning. The right pupil was dilated, the left contracted, neither reacted to light. Pulse 60, irregular. The right arm was slightly stronger than the left. Paresis of the right side of the face. There was some bogginess over the left parietal. She was trephined over the left side but nothing found. At the subsequent post-mortem examination, there was two and a half ounces of clot subdurally on the right side, and several small perivascular hæmorrhages into the pons. Extensive bruising of the brain was present but no fracture of the skull.

A somewhat similar case was under Mr. Davies Colley's care in 1889 (Report 307).

An unknown man was knocked down by a van and admitted to the hospital. He was unconscious and with a slow pulse on admission. A small scalp wound above the occipital protuberance was present. After he had been in a few minutes the pulse fell to 40, the pupils became unequal, the left contracted and right dilated; some rigidity was present on the left side, including the facial muscles. No strabismus was present. He was trephined over the scalp wound, but the exposed membranes found to be healthy. At the autopsy, he had an ounce of blood subdurally, bruising of the brain, and multiple hæmorrhage into the pons.

In cases where the subdural hæmorrhage is probably due to a cerebral hæmorrhage breaking through externally, a very guarded prognosis will be given, even though the patient may survive (as in case 70) and in those cases where there is found thickening of the meninges, associated or not with hæmorrhage (the so-called pachymeningitis interna hæmorrhagica) the patient, though he recover, will always be liable to serious trouble following slight injury. In the Medical Record for April 30th, 1898, p. 115, Dr. R. Van Santvoord reports a case of subdural hæmorrhage found at post-mortem examination where there was "general congestion of the meninges with thickening of the pia," concerning which he says, "this man evidently had a chronic alcoholic meningitis before his injury. The essential factor in his case was apparently a traumatic exacerbation of this disease." In the American Journal of the Medical Sciences, vol. 109, pp. 404-406, there is a case of "Localised hæmorrhage beneath the pia mater, over the upper third of the Rolandic area, due to a fall on the head." By Dr. J. J. Putnam.

The patient was a lady, aged 72, who fell down some steps. She was not fully stunned and with assistance walked into the house and upstairs. She was seen by Dr. C. P. Putnam and then appeared in a good general condition, but had bruising of the scalp. For twenty-four hours she did well, after which spasms, followed by paresis and ultimately paralysis set in. Two days later coma was present. Death took place ten days after the accident. At the autopsy, there was no fracture of the skull but subdural hæmorrhage on the right side of the skull, especially posteriorly. Microscopic examination showed the existence of an old vascular false membrane. It was from these vessels that the subdural hæmorrhage had occurred.

In the Transactions of the Clinical Society, vol. xxv., pp. 157-160, is reported a case of "Pachymeningitis Interna Hæmorrhagica treated by trephining." By Mr. Stanley Boyd.

An innkeeper, 40 years of age, accustomed to live freely, fell on March 31st, 1891. Bruising of the scalp with a somewhat dazed condition, was found soon after. For the next fortnight he suffered from headache and remained in bed. He went back

to work on April 21st. Two months later, about June 18th, headache and paresis set in. When seen by Dr. Mitchell Bruce on the 22nd the discs were normal. Right sided paresis developed into paralysis, and on the 28th he was comatose, the pulse was quick and full. A diagnosis of abscess on the right arm area was made and the patient trephined on the left side. The dura was bulging and motionless. A flap of the dura mater was turned down. It was adherent on the deep surface but stripped off easily, leaving exposed a slightly bulging, motionless, greyish-yellow surface. This required a distinct amount of force to send the knife through it. When opened four ounces of dark red clear fluid escaped. "Evidently a cystic clot had been opened." A drainage tube was left in the cavity. Recovery was slow. Mr. Boyd suggests that the slow recovery of consciousness may perhaps have been due to rigidity of the cyst wall, preventing expansion of the brain. There was no albumen or sugar in the urine before operation. He went to the seaside on August 15th, and in May of next year was very well.

This case is particularly interesting in that a long latent interval was present, during which time, from the condition of the clot, the blood was present in the cranium. It is extremely probable that the case reported by Taylor and Ballance passed through the stage found here before finally becoming the typical cyst removed at operation. It had originally been my intention to deal fully with the remote history of cases of subdural hæmorrhage, which pass on to organisation of the clot and the formation of cysts, but this paper has already reached a length to which, in the first place, it had no pretensions, so that beyond giving a list of the reported cases which I have found, I shall dwell no more on the subject. There are three other points of some importance to be considered, however, before leaving prognosis, viz. :—

1. The question of perfect recovery from paresis or paralysis.
2. The relationship of traumatic subdural hæmorrhage to traumatic epilepsy ; and
3. The relationship between traumatic subdural hæmorrhage and insanity.

1. With regard to the persistence of paresis or paralysis after operation and removal of the clot, it may depend upon either the damage produced by continuous pressure or the result of laceration of the brain. The following facts have a bearing upon the subject. In case 10, the operation was performed thirty-six days after the injury (but only twenty-one days after the onset of symptoms) with perfect recovery. In the case reported on page 104, where compression of the brain had been present probably for six weeks, and where, after removal of the cerebro-spinal fluid, the brain in the Rolandic region was over one inch from the skull, the mother wrote five months later to say that the child plays about with other children, and she considers her perfectly recovered. In vol. xviii., p. 379, of the Transactions of the Medical Society is the report of a case where hemiplegia followed injury two years before. At operation, a cyst of the arachnoid was removed, after which freedom of movement in the hitherto rigid limbs followed. From these cases it would appear that as long as the brain surface is uninjured, a very long period of compression may occur, and yet if it be relieved perfect recovery follow.

On the other hand, it will be seen that in the case reported by Dr. McBurney some paresis persisted. This may be considered with the case reported by Dr. Hale White, published in the Pathological Society's transactions (vol. xxviii.)—"Old meningeal hæmorrhage with softening and secondary descending degeneration."

Mary G., æt. 36. Seven years ago fell down whilst skating. Eighteen months ago the right arm and leg gradually became weak and she completely lost the use of these limbs, but after four months could walk again with assistance. She gradually sank and died from a fæcal fistula in connection with an old femoral hernia. Autopsy.—Cranial bones and cerebral arteries normal. Left side over ascending frontal and ascending parietal convolutions, back part of a little of the first and third frontal and posterior third of second frontal was some soft yellowish-white material, evidently very old blood clot. The grey matter corresponding to this area was gone, there being a distinct

depression in the brain substance filled up by the above material. The white substance beneath was little altered. No distinct degeneration could be seen in the internal capsule or pons, but the pyramidal tract in the medulla and the crossed and direct tracts in the cord were decidedly grey and translucent and hard; the lateral columns on the affected side were certainly the smaller. The kidneys were granular.

In both these last two cases some laceration of the brain may have been present, and I think from the reported cases that it is justifiable to say that, if at the time of operation the brain is not lacerated, if the patient recover, his or her paresis or paralysis should entirely disappear. In the event of some laceration associated with paresis or paralysis being present, and especially if the paresis or paralysis has persisted for a long time, some permanent damage may remain.

With regard to the relationship of traumatic subdural hæmorrhage to traumatic epilepsy and insanity, from the reported cases and opinions it seems very improbable that this form of hæmorrhage leads to persistent fits or ultimate insanity. In the case of fits occurring as the result of pressure by clot, it seems almost certain that when they occur they progress rapidly and lead to early operation, very often a diagnosis of some purulent collection within the skull¹⁶ being made; whilst it is a truth, I think, that the hæmorrhage found subdurally in lunatic asylum autopsies is not the cause of the insanity but is the result of the atrophy of the brain associated with the mental change. I would draw especial attention on this point to Dr. Wigglesworth's paper on "Hæmorrhages and False Membranes within the cerebral subdural space occurring in the insane (including the so-called Pachymeningitis)."¹⁷

¹⁶ See Walsham "On Trephining the Skull in Traumatic Epilepsy," St. Bartholomew's Hospital Reports, Vol. xix.

¹⁷ See also Vol. xliii. of the Pathological Society's Transactions. "Subdural Hæmorrhage," by E. T. Wynne, M.B., also Dr. Newton Pitt's paper "Double Subdural Cysts of Hæmorrhagic Origin," in the same volume. In the forty-sixth volume of the same Society's Transactions is a paper by Dr. C. F. Beadles on "Some Gross Lesions in the Brains of Lunatics, etc." See also "Brain," Vol. xv., "The Traumatic Factor in Mental Disease," by Dr. W. J. Mickle.

Finally, on the uncertainty of the future history of any case of head injury, I may quote a sentence from Gross' *System of Surgery* (1882)—"The prognosis of wounds of the brain and its membranes is too variable to admit of general specification. While in some cases, indeed in a great many, the slightest injury causes death, in others, attended with excessive shock and the loss of a large quantity of blood and cerebral matter, the most prompt and satisfactory recovery occurs."

In conclusion, I would thank the surgeons of Guy's Hospital for permission to publish the twenty-four hitherto unrecorded cases which are derived from the Surgical Reports of Guy's Hospital. These twenty-four cases number a third of the total cases upon which this paper is based.

To Mr. Jacobson and Mr. Symonds, surgeons of Guy's Hospital, I owe a special debt of gratitude. Were it not for their assistance and advice this paper would, even if written, never have approached the completeness which it ultimately endeavoured to realise. To anyone reading it, the numerous references to Mr. Jacobson's paper on "*On Middle Meningeal Hæmorrhage*," are a sufficient indication of my indebtedness to this work, but I may say that this is only a small part of a greater series of obligations, the extent of which I alone can realise and which I take this opportunity of very gratefully acknowledging.

ABSTRACT OF CASES.

CLASS A.

No. 1.—Mr. Golding-Bird and Mr. Dunn. *Guy's Hospital Surgical Reports*, 1901. Report No. 586.—Walter A., æt. 24, was knocked down (November 1st) by an engine towards which his back was turned. He was rendered unconscious and brought up at once to the hospital. On admission, he was unconscious; there were three scalp wounds, two over the vertex, and one in the occipital region. There had been very little blood lost, and all bleeding had ceased. The tenth rib was broken, and there was a large oval bruise on the back. The wounds were cleansed and stitched up, gauze drains being used, no anæsthetic being necessary. On November 2nd, the wounds were healthy, but he had severe headache and was sick during the morning. He got up after tea, but was glad to get to bed again. On the 4th, he still complained of severe pain in the frontal region, and slept badly. On the 5th, the headache still persisted; the wounds were quite healthy. On November 6th, there is a note saying he did not complain of any pain and was discharged. The man was readmitted to the strong-room on November 16th, *i.e.*, ten days after his discharge. The history was that since leaving the hospital he had done no work, had spent most of his time indoors, complaining of aching pains in the back of his head, and had been restless, very irritable and unable to sleep. He took food well, but the bowels were constipated. Three days before readmission the pains in the head became much worse and the patient excessively irritable. On the day previous to readmission he developed a squint and soon became totally blind, began to lose the power of speech and stammered. At times the right arm was paralytic, and at times rigid. There was no vomiting. On readmission the man was excessively restless, but inclined to be drowsy. Pulse 76. Respiration 20. Temperature 99°. Aphasia was marked, and movement of the right arm impaired. It is said that although the patient could see objects presented to him he did not appear to recognise them. Hearing was apparently normal. He was seen by Mr. Dunn and Dr. Fawcett, who diagnosed a clot pressing on the arm area and decided to trephine at once. Operation: A.C.E. mixture being administered, the dura mater was exposed and seemed dark brownish-blue in colour. It was incised and reflected exposing a small blood clot and some dark-coloured blood which slowly welled up. This was carefully removed with the aid of a blunt spoon. The piece of trephined bone was replaced and the skin sutured continuously. November 17th: Patient appears much better this morning. Temperature 98. Pulse 80. Respiration 16. Delirious during the night, and this morning complains of headache, but he speaks quite well, although slightly indistinctly. Movement in the right arm appears quite normal. There is no sickness. Urine slightly alkaline, sp. gr.

1022, no albumen, pus or sugar. On November 29th, when he was discharged, he seemed perfectly recovered, having full power of movement in the right arm and free speech.

No. 2.—Mr. Jacobson. Guy's Hospital Surgical Reports, 1901. Report No. 566.—Thomas M., æt. 62, was pushing a trolley up a raised platform (December 9th, 1901) when he fell a distance of three feet to the ground, striking the right side of his head against a stone. On admission, he was unconscious, with a scalp wound above the right ear. Pulse regular, fairly full and strong. Temperature 99°. Right pupil smaller than left. Consciousness was present next day, but the pupils were still unequal. On the 12th, he was restless and trying to pull his bandages off, but from this time up till the 24th he steadily improved. From the 24th to 28th he gradually became comatose, and on this last date his breathing becoming stertorous and his condition critical, it was decided to operate. No anæsthetic was administered. A flap being turned down in the right temporal region, the skull was trephined and the dura exposed nonpulsating. Incision of the membrane exposed a quantity of dark semi-fluid clot, which was removed. Cerebral pulsation now recommenced, causing more of the clot to collect. A gauze drain was inserted. The patient died at 9.10 p.m. on the 30th, remaining unconscious till the end.

No. 3.—Mr. Davies-Colley. Guy's Hospital Surgical Reports, 1898. Report 341.—John F., æt. 56, fell from a railway platform and struck the back of his head on a dummy capstan (July 2nd). He did not lose consciousness, and after having a scalp wound dressed was sent home. Next day he could not use the left arm or left leg. This paralysis persisted until his admission into the hospital on July 8th. On admission, there was bare bone exposed at the bottom of a wound in the occipital region. There was paralysis of the left lower limb, and paresis of the left upper. No loss of sensation. No facial paralysis. Pupils equal and react. Answers questions intelligently. Pulse 80, regular, rather easily compressible. Temperature 99.2°. Respiration 18. The patient remained much the same, but his restlessness required the administration of soporifics at night until the 19th, when, as he was growing weaker, and no improvement had taken place in his paralysis, it was decided to operate. The trephine was applied over the middle of the ascending frontal convolution. The dura mater appeared black. Blood was exposed subdurally and removed. The bone being replaced, the wound was sutured. From the time of completion of the operation he made no improvement, required sleeping draughts for his restlessness and insomnia, and becoming weaker and weaker he ultimately died on August 5th. At the autopsy, only the brain was examined. A clot was found with a depression in the brain a short distance in front of the upper part of the fissure of Rolando.

No. 4.—Mr. Bryant. Guy's Hospital Surgical Reports, 1885. Report No. 274.—Jessie S., æt. 48, found insensible and bleeding, by the police. Said to have fallen whilst drunk (November 28th). On admission, she was insensible, and could not be roused. Pupils equal, moderately dilated and not reacting to light. Deep scalp wound over right side of head two inches long, with exposure of bone. Pulse rapid and feeble. Four hours after admission she had a fit. Some weakness of right side of face and right arm noticed.

Epileptiform convulsions now occurred frequently, affecting the left side at first, but later becoming bilateral. The fits were kept in check with bromides. On the 29th she could be roused, and answered her name. The fits persisted all day save when restrained by bromides. The pulse increased in rate, and she died on the 30th, at 5.30 a.m., Cheyne-Stokes' respiration occurring towards the last. Her husband said she suffered from epileptic fits, and was a heavy drinker. At the autopsy, no fracture of the skull could be found; there was no extradural hæmorrhage. Middle meningeal artery uninjured. Over the left temporo-parietal part of the brain compression had been produced by two ounces of blood, situated beneath the dura mater. The clot was easily removed, and no laceration of the brain discovered. There was no intracerebral hæmorrhage. The vessels of the base showed some thickening, and rather opaque appearance. They were also distinctly pouched in one or two situations. Heart normal. Liver fatty. Right kidney atrophied, and contained two cysts. Left kidney hypertrophied, weighing twelve ounces, also fatty and mottled, with adherent capsule in places.

No. 5.—Mr. Mansell Moullin. *Clinical Society's Transactions*, vol. xxvii. "Traumatic arachnoid hæmorrhage with symptoms on the same side as the lesion" (November 10th, 1893).—T. C., a stevedore, æt. 43, had a violent blow on the side of the head with the hook of a crane at 10 a.m. Did not lose consciousness and continued his work until nearly 1 p.m., when he felt faint, and quickly became unconscious. On admission, the breathing was deep and noisy. Pulse 70, full and regular. Temperature 99°. Weakness of right face muscles, rigidity of right limbs. Pupils evenly contracted, but after exposure the right pupil dilated more than the left. The right side of the skull was exposed in the hope that a fissured fracture might be found extending round to the left side from the site of injury, but none was found. Next day there was some improvement, and towards evening he recovered consciousness. No increase in paralysis. For the next two or three days there was no material change, then delirium at night, coming on at intervals, set in. Nine days after the accident, as coma was deepening and the breathing becoming more and more stertorous, the left side of the cranium was explored. The dura mater did not bulge on exposure. It was incised. The brain bulged at once and began to pulsate. Bone replaced. Death occurred twenty-four hours later, the temperature rising from normal to 109° just before death. Autopsy: no trace of fracture. No extradural hæmorrhage. "Under the dura on the right side, between it and the arachnoid, was a large black clot covering the greater part of that hemisphere. Nearly three-quarters of an inch thick over the lower end of the fissure of Rolando, and from there it thinned off in all directions, but it reached nearly to the falx above, on to the occipital lobe behind, and to the clinoid process below." The brain was cupped, but uninjured. There was no atheroma. There were strong reasons for believing that the source of the blood was the middle meningeal artery.

No. 6.—Mr. Raymond Johnson and Dr. Risien Russell. *Clinical Society's Transactions*, vol. xxxiv., case 26. "Traumatic subdural hæmorrhage occasioning convulsions on the 6th day after injury, and successfully treated by operation (May 24th, 1901).—Frederic W., æt. 23, admitted to University College Hospital early in the morning of October 23rd, 1900, suffering from

frequently recurring epileptiform convulsions affecting chiefly the left side of the body. On October 17th he had fallen down some steps whilst drunk, and being picked up unconscious had been put to bed. Next morning he walked to a medical man and had a scalp wound dressed. He complained of headache and feeling shaken. He did no work, but kept free from any definite symptoms until the night of October 22nd at 10 p.m., when fits set in. On admission to hospital, he was bathed in perspiration. Pulse 100. Temperature 102° . Each fit lasted two to four minutes, and the intervals between the fits varied from one to five minutes. The onset of a fit was marked by his turning head and eyes to the left, or by twitching of the left side of the face. The left side and right leg, and occasionally the right arm, were affected. The spasms were clonic. In the intervals he was sufficiently conscious to answer questions. Pupils equal, medium size and reacted. No facial paralysis. Left arm weaker than right. Chloroform was administered to stop convulsions. Bromide and chloral also administered. The urine contained a dense cloud of albumen. Uræmia and idiopathic epilepsy were both seriously considered, but meningitis was finally considered to be present (for discussion of diagnosis see under diagnosis in text, pp. 92 & 93). Operation was decided on. The skull being opened, the dura mater bulged and had a bluish tint as though blood was beneath it. On incising this membrane, two to three drams of blood escaped. The brain appeared perfectly normal. The dura mater was sutured, the bone *not* replaced. There were no fits after operation, but it was nearly a month before the left arm completely recovered. The temperature rose to 105° four hours after operation, and for four days it varied between 101.4° and 104° . The patient passed through a mild attack of delirium tremens.

No. 7.—Professor McBurney. *Brain*, vol. xiv., p. 284.—A physician, æt. 40. Thrown from his carriage August 17th, 1889. Slightly stunned by the fall, but with no wound, and able to assist his wife. For several hours he seemed only suffering from bruises and did some professional work. In the course of the evening, however, he became delirious, then stupid, and, for the next three days, lay in a semi-comatose condition. On the morning after the injury he was found hemiplegic on the right side, and aphasic. When after a week his consciousness had fully returned, it appeared that the aphasia was purely motor. He came under the care of Professor McBurney at the Roosevelt Hospital in December, nearly four months after the accident. He then had partial right hemiplegia with motor aphasia, was mentally unstable, and the left pupil was one-third larger than the right. There was no facial paralysis, and the optic discs on examination were quite normal. Professor McBurney came to the conclusion that a vein had ruptured and slow hæmorrhage occurred over the left hemisphere at the posterior part of the third frontal convolution and over the anterior central convolution in its middle third. On December 13th, he trephined the skull over this region; the dura mater did not pulsate. On opening it the pia was found very cedematous and discoloured, and the surface of the brain separated from the dura by a space of half an inch in depth and did not pulsate. Blood clot was seen lying beneath the pia mater. The clot was removed. Drainage with tubes was employed for a week. Two months after operation he walked with a cane. The return of speech was slow but continuous. Fifteen months after operation paralysis of the right hand remained. This was thought to be due to permanent nutritive changes in the arm area.

No. 8.—Mr. T. Chevalier. *Medical and Physical Journal*, vol. viii., p. 505 (1802).—A baby, 1½ years old, fell out of the servant's arms at 10 o'clock in the forenoon and struck its head violently against the head of a door. Taken up senseless. After remaining comatose for one and a half hours, the infant went into convulsions. After these had continued for two hours Mr. Chevalier was called in. There were no external marks of injury on the scalp but the fontanelle was distended, and a bleeding vessel under the dura mater was diagnosed. An angular incision, through the fontanelle, was made on the right side, since the limbs on the left side were much more violently convulsed than those on the right. Blood was seen under the dura mater, which was punctured. The blood spurted out one foot: three to four ounces at least were discharged. It appeared venous. All spasms ceased in three-quarters of an hour, and the baby made a perfect recovery.

No. 9.—Mr. Ogle. From B. Brodie's Paper in the *Medico-Chirurgical Transactions*, vol. xiv.—A woman, whose age is not given, had fallen down into a cellar from the street head foremost. Picked up comatose. Shaving of the head was ordered. There was no wound of the scalp, but she flinched when pressure was made on a spot near the anterior superior angle of one of the parietal bones. An incision was made here, but no fracture discovered. Bone being removed with a trephine, the dura mater of a dark colour rose into the opening nearly as high as the external surface of the cranium. The dura mater was punctured with a lancet. Blood spurted out to a height of some feet. The woman who till that moment had remained totally insensible regained consciousness and spoke, describing the accident. She recovered without any untoward symptoms.

No. 10.—Dr. Otto G. T. Kiliani. *Annals of Surgery*, 1901, 33, p. 325.—A man, age not stated, admitted to the German Hospital on June 6th, 1900, with a history that on May 5th he was struck by a falling brick (forty feet) which, as he wore a Derby hat at the time, neither caused a wound or produced unconsciousness. He was dazed for a short time. On May 21st he was seized with violent frontal headache. On the 26th, he became dizzy and nearly fell whilst walking. On June 1st hesitancy of speech set in. On admission to hospital he was somewhat somnolent, becoming slightly restless when aroused. Slight spastic paresis of right arm and hand, and to a less extent of right leg. Ataxia in both arms. Partial motor aphasia. No facial paralysis. Optic discs normal. Temperature 98° to 100°. Pulse ranging from 68 to 92 (mostly 64 to 68). Respiration 12 to 24. June 11th (thirty-six days after injury), anæsthetic administered and a flap turned down on the left side of the skull. Dura mater exposed, after removal of bone, nonpulsating and with large tensely-filled veins of a dark colour shining through. Incision into dura mater and evacuation of the clot, which was an inch and a quarter thick, covering practically the entire left hemisphere. Pia mater and convolutions normal. The wound was drained with a tube. A perfect recovery followed, and on August 19th he is stated to be perfectly well and attends to business.

[Dr. Kiliani also refers to a case reported by Hahn, *Ein Bertrag zur Chirurgie des Gehirns*; *Centralblatt für Chirurgie*, 1896, 6; *Deutsche Medizinische Wochenschrift* 14-16; Subdural large hæmatoma without fracture. Trephining. Recovery.]

No. 11.—Drs. Russell and Pinkerton. *British Medical Journal*, i., 1895, p. 1318.—J. H., a healthy, wiry man of 67, was thrown from his tricycle on March 3rd, 1894. He was not stunned but had wounds which bled freely. On March 4th, when first seen, he had vomited all that he had taken. On the 5th aphasia set in with paresis of right arm. At 11 p.m. on the 6th convulsions set in, affecting the whole body. Three such convulsions were noted. On the 8th and 9th his condition was improved, and he was conscious, but from the 10th to 12th he gradually got worse, became first lethargic and restless and finally comatose. Pulse 60, full. An operation was performed on March 13th. After two trephine openings had been made a point of "increased resistance" was found. The dura mater was incised here and black clotted blood exuded with each pulsation. This was removed and the cavity drained. The man moved the right arm a little immediately after the operation. The aphasia began to improve on the fifth day. Advancement was slow until the eleventh day and then rapid, so that by the twenty-fifth day he could speak fairly well.

No. 12.—Dr. Barlow, (quoted by Sir Samuel Wilks in his paper on "Sanguineous meningeal effusions: spontaneous and from injury").—A. G., æt. 18, admitted into Guy's Hospital under Dr. Barlow on July 17th, 1856, and died next morning. He received a blow on the head whilst fighting in a barge on July 6th. He did not suffer much in consequence, and continued his employment during the next ten days, but on the 17th he had headache, and it is said that he evidently had considerable pyrexia on admission. Post-mortem (Report No. 137 for 1856): There were no external signs of injury to the head and no fracture of the skull. On the right side the dura mater was flaccid and purple. Four ounces of blood, either fluid or in the form of loose coagulum, were present subdurally on this side. The blood compressed the right side of the brain and had extended to the base, compressing the Pons Varolii. The left hemisphere was unaffected. There was no cerebral laceration, or source of hæmorrhage found. No laceration of sinuses or meningeal arteries. An examination of the clot seemed to show that there was an old part becoming organised, and a more or less recent hæmorrhage in addition. All the internal organs were healthy.

No. 13.—Dr. Goodhart. *Guy's Hospital Reports*, vol. xxi. From paper on "Meningeal Hæmorrhage." (Case 31.)—James R., about 35. Struck by another man twice on the head. Brought into the hospital dead. At the post-mortem examination there was slight ecchymosis in the upper part of the right temporal muscle. No fracture of the skull. About the brain in the arachnoid and subarachnoid space there was a quantity of blood which had gravitated to the base. The subependymal arteries were fatty here and there. Many recent small extravasations into the lungs.

No. 14.—From the St. Bartholomew's Reports. Cases from Mr. Walsham's Wards. (No. 1922. Kenton.)—Man, æt. 37, admitted unconscious. He had been knocked down by a cab the day before, the wheel, it was said, having passed over his neck. The man was very drunk, and was washed out in the surgery. Soon became noisy and delirious and eventually drowsy, whilst paresis of the left side of the face, the left arm, and to a less extent of the

left leg set in. During the night a fit (thirty seconds) occurred, followed by general twichings and afterwards a complete paralysis of the left face and arm, and increasing weakness of the left leg. Trephining over the right motor area was performed 12.30 p.m. No extradural blood was present, but on incising the dura mater a large quantity of blood escaped. The patient slowly recovered consciousness, and in two weeks the paralysis had disappeared and the temperature fallen to normal. From this time he gradually recovered, and left the hospital quite well.

No. 15.—From the St. George's Hospital Reports (1875). Cases from Mr. Holmes' Wards. No. 467.—John T., æt. 60, fell twelve feet, pitching on his head. Admitted shortly afterwards with slight concussion, from which he was easily aroused. Starred wound over right frontal eminence, but without exposure of bone. Twenty-four hours after admission he gradually lost consciousness and became restless. Twitchings in the muscles of the face and the left upper extremity followed. Within three-quarters of an hour of the onset of these symptoms the wound in the forehead was enlarged and the bone examined. No fracture was detected. Twitchings with unconsciousness continued, the patient dying quietly fifty hours after admission. The breathing became stertorous half an hour before death. At the autopsy, a large, recent coagulum was found on the right side of the brain, in the cavity of the arachnoid, also slight extravasation of blood in the neighbourhood of the veins of Galen. The first part of the aorta was atheromatous and dilated, the kidneys granular and cystic.

No. 16.—Mr. Henry Morris. *Lancet*, November 11th, 1882.—James J., æt. 31, was admitted into the Middlesex Hospital, December 14th, 1881. For a fortnight before admission he had been living in a dissipated condition at Brighton, and on his return to London, had fallen heavily, striking the back of his head against the kerbstone. Being picked up half unconscious, he was first taken to a police station, but subsequently to the hospital. On admission, he smelt strongly of drink, and was in a muddled state, with a small scalp wound slightly to the left of the occipital protuberance. The periosteum was not exposed. Pupils equal and reacting. Respiration and pulse normal. No signs of any fracture of the skull. Stupidity put down to alcohol, and passed off in several hours. For the next three days the only abnormal feature of his case were complaint of headache and double vision. He then passed through an attack of delirium tremens, from which he had recovered on December 22nd, when he seemed quite well though weak. On December 25th he went to church, but complained of feeling drowsy and having pain in his back. During the night the nurse on duty noticed how restless he was, and next morning (26th) it was found impossible to wake him. At 10 a.m. he was found to be in coma, with the right pupil widely dilated and the left very contracted; neither responded to light, and the conjunctivæ were insensitive. Pulse 100, small and jerky. There was very slight twichings of both upper limbs, but no paralysis at all. A sample of urine (which had been free from albumen on the 17th) was found to contain a lot of albumen. Temperature 101.6°. There was no œdema round the scalp wound, but a little purulent discharge from it. He was treated with pilocarpine and calomel, and on the 27th seemed better, answering when spoken to,

but the pupils remained unequal. The urine was still highly charged with albumen, so that a hot-air bath and jalap were ordered. On December 28th and 29th, he was much the same, the albumen, however, being half what it was. Later his breathing became more stertorous, and he could neither speak nor swallow. At 9 p.m. on the 29th his temperature rose to 104.2°, and at 3.10 a.m. on the 30th the man died. At the post-mortem examination made by Dr. Fowler, there was no fracture of the skull. The right half of the dura mater covering the brain appeared somewhat distended. Blood was found spread out beneath it. This was most abundant over the frontal lobe and in the anterior fossa, but there was also a considerable quantity beneath the parietal eminence, and over the occipital convolutions. The blood was of a mahogany tint, the coagula being soft and adherent to the under surface of the arachnoid. The source of the blood could not be found. There was a small clot on the left side immediately below the wound. There were some spots of atheroma in the aorta, the lungs were emphysematous, the kidneys were large and the capsule in places adherent, but beyond swelling of the cortex, probably from slight infiltration, there was no evidence of disease.

No. 17.—Reported by Mr. Leopold Hudson, in the *Lancet* of March 8th, 1890. The case was under Mr. George Lawson.—J. W., male, æt. 20, single. The patient, whilst boxing, was noticed to become suddenly weak, but continued to box for a few seconds, when he fell back across the ropes. He did not strike his head as he fell, but when picked up he was quite insensible. As he did not recover consciousness at the end of half an hour, he was taken to the Middlesex Hospital and admitted. No particularly hard blows had been given. On admission, he was comatose, the pulse was 50, full, the respiration stertorous and interrupted, the pupils fixed and dilated, surface of the body cool, the extremities powerless and flaccid. The urine (by catheter) contained no albumen. An ophthalmoscopic examination was negative. The patient lived for two hours after admission. No spasm was observed at any time. At the autopsy (made by Dr. Sidney Martin) a little dried blood was found in the anterior nares. A subcutaneous ecchymosis was present below and to the outer side of the left eye. No fracture of the cranial bones was present. No injury to the dura mater or meningeal arteries was present. On opening the dura mater there was found over the middle lobe of the left cerebral hemisphere a quantity of soft blood clot depressing the subjacent convolutions. It appeared to be derived from the arachnoid. There were three very small ecchymoses beneath the pia mater on the left side. No laceration of the brain was present. With the exception of slight atheroma in the commencement of the aorta the viscera were normal.

No. 18.—Mr. Edmund Owen. *British Medical Journal*, 1888, vol. ii., p. 817.—Wm. MacC., 9 years old, was brought to St. Mary's Hospital on May 8th, stunned by a fall from a cart. He vomited profusely, turned his head from the light, and when asked questions said "go away." Pupils equal and reacted. No fracture could be detected. The movements of the limbs were perfect, but he was very drowsy. Next day he was restless, muttering and jumping out of bed. Temperature 101°. Five days after admission Jacksonian epilepsy set in, affecting in the first place the right facial and suprahyoid muscles, and later the muscles of the right hand,

causing clenching of the fist. The evening temperature was 101.8°. During the next day he had slight fits, but towards evening a most severe one affecting, in addition to the muscles previously mentioned, the diaphragm and right sterno-mastoid. The boy was aphasic, but conscious. On May 15th at 4 p.m. ether was administered and the skull opened. The dura mater presented a somewhat greenish hue and bulged. On incising it a quantity of dark fluid blood escaped. The total blood removed was thought to amount to half an ounce. It lay over the lower part of the left motor area. The space under the dura mater was irrigated with warm carbolised water, and the dura sutured. On the 16th and 17th, the fits recurring, further extravasation was diagnosed. On re-operating a small quantity of black fluid blood was removed from the subarachnoid space, which had not previously been opened. He was discharged cured on June 15th.

No. 19.—Dr. J. S. Horsley. *Medical News* (Philadelphia), November 3rd, 1894.—A. B., coloured, aged 43, the subject of chronic syphilis and a hard drinker, but with no previous history of epileptic fits or cerebral lesion, fell off a waggon on August 6th, 1894, striking the left parietal eminence against the kerbstone. A wound, one to one and a half inches long, not reaching bone, was formed. He got up, and seemed to have a slight convulsion of his left arm. After some attention he was sent home (4.30 p.m.). At this time he was quite conscious. Half an hour later a doctor was sent for and found the man having general convulsions. He gave morphine and atropine, and the spasms became localised to the left arm. At 6.30 p.m. the man was completely unconscious, with stertorous breathing, a full slow pulse, and slightly elevated temperature. Four hours later he was much worse, with shallow respirations, slightly dilated pupils, and a weak pulse with a rate of a hundred and twenty-five per minute. There was spasm of the left arm. The head and eyes were rotated to the left. He died at 12.30 p.m., no operation being performed on account of his adverse surroundings. Post-mortem: No fracture. Subdurally on the outer side of the right temporo-sphenoidal lobe was an irregularly-shaped clot. The source of hæmorrhage was an artery between the middle and inferior temporo-sphenoidal convolutions. The clot was about two inches at its broadest diameter, one inch at its widest, and one-sixth of an inch at its thickest point. The walls of the blood vessels seemed degenerated.

No. 20.—From the *Boston Medical and Surgical Journal*, vol. cxxx., p. 201. (1894.)—A. H. L., a tall athletic student, 19 years of age, was struck in a friendly sparring bout on the left jaw. Large gloves were used. The patient, after commenting on the excellence of the blow, said he felt queer, put both hands to his head, jumped up and down several times, reached out his hands for the wall, staggered, and fell unconscious. This was at 5 p.m. on February 13th. Consciousness was not recovered, breathing became stertorous, pupils first dilated then contracted. Within four hours rigidity had appeared in all four extremities, preceded by restlessness. At one a.m. on February 14th a consultation was held as to the advisability of operation. At this time all four extremities were in a condition of rigid contraction. There were in addition clonic movements of the right hand. Eyes turned somewhat to the left but without strabismus. Both pupils large and

only slightly reacting to light. Breathing heavy, abdominal, and with a tendency to Cheyne-Stokes' respiration. Tendency to lateral nystagmus. No priapism. Pulse 96, strong, full. Temperature 101°. No signs of fractured base. The signs of pressure on the pons and medulla were considered to contraindicate operation. On the 15th the rigidity entirely disappeared leaving all the extremities paralysed. Temperature 102°. Respiration 30, stertorous. The patient gradually failed and died quietly at 3.50 p.m. on the 18th. Temperature in afternoon 106°. At the autopsy there were no signs of external injury. The surface of the brain was covered with extravasated blood beneath the dura mater. The extravasation was most marked over the occipital lobes and on the left side. The blood was blackish in colour and of a tarry consistency. On removing the brain the middle fossa on the left side was found to contain several ounces of the same black tarry blood, which also extended into the vertebral canal. There was a small rent in the lateral sinus near the outer margin of the temporal bone. About this rent for a short distance the dura mater was dissected up. There was no fracture of the skull, and no extravasation of blood into the brain.

No. 21.—Dr. F. L. Wells. Medical Record (New York), vol. xli. (1892).—The patient, a German, æt. about 23, was walking along the street when he was suddenly seen to fall and become convulsed. These convulsions ceased before the arrival of the ambulance, but were described as being of his face and all his extremities, associated with frothing at the mouth and blueness of the face. On admission to hospital he was entirely unconscious, tongue bitten, and no evidence of injury save a scar on the left side of the head anterior to and a little above the ear. It was judged to be three to six weeks old. Both pupils were dilated, but reacted to light. Temperature 102°. Respiration 30. Pulse 120. No paralysis. Urine of specific gravity 1020, with a slight amount of albumen present, no casts and no sugar. Convulsions occurred at intervals of a half to two hours, were general, and lasted one to two minutes. In the intervals between the fits the patient was completely unconscious, delirious, and with difficulty kept in bed. He was frequently given morphine sulphate hypodermically. Towards evening the temperature rose to 104°. Next day, August 21st, the temperature was 103°, the pulse rate 110, and the respirations 26. The convulsions were carefully noted and were found to begin in the muscles of the right eye or right corner of the mouth. They advanced then to the right arm and leg, and then became general. The advance was so rapid, however, as to make them appear general from the first. The evening temperature was 105°. It was followed by sweating. The convulsions became more severe and more frequent. During the night following the convulsions were nearly constant. It was decided to operate. Before operation the temperature was 105°, pulse 123, and respiration 22. At the operation a flap was turned back including the scar. The periosteum was found more firmly adherent than is natural. No evidence of fracture could be detected, and no elevation or depression existed. The skull was trephined at about the centre of the motor area. The circle of bone was removed with great difficulty, the dura mater being so adherent as to tear away pieces of the bone. The bone itself was three sixteenths of an inch thick, and perhaps a little more vascular than normal. No depression of the inner table was found, but the dura mater was dark in

colour and nonpulsating. A crucial incision was made in the tense membrane when blood clot immediately protruded. The incision was then enlarged and a handful of clotted blood removed which was covering the motor area of the face, arm and leg; the largest part being over the face centre. Microscopical examination showed the clot to be undergoing softening and a transformation into pus. The pia mater was seen to be very much reddened and inflamed. The brain itself was greatly compressed but not softened. The cavity was cleansed with biniodide, 1 in 20,000, and iodoform gauze used for drainage, the bone not being replaced. The patient, on coming round from the ether, spoke the first rational words since admission, asking for a drink of water. Temperature at night (four hours after operation) 103°. During the night following and the 23rd several general convulsions occurred. On the 26th, the temperature fell to 99°, and kept at about that level until his recovery. He gradually recovered and gave the following history: That he was struck on the head three weeks before admission to the hospital, the scar found on admission being due to the cut then received. He had never been troubled by the injury until a fortnight after the blow, when severe headache set in, and for a few days before admission he noticed that the right arm and leg were not as strong as usual. He was discharged September 23rd.

No. 22.—Dr. S. T. Armstrong. *Journal of the American Medical Association*, June 18th, 1887, vol. viii.—George J., æt. 53, a shoemaker, was, on February 27th, struck on the left forehead by a brick. An irregular lacerated wound about half an inch above the outer edge of the eyebrow was caused. The man was rendered unconscious for a short time. He was seen by Dr. Armstrong next morning, who treated his wound. He walked half a mile to have the required dressings, and was discharged with the wound healed on March 14th. On April 24th (i.e., six weeks after his first discharge) he consulted Dr. Armstrong again for a roaring in the head which had persisted since the injury, and on April 18th he noticed that there was a tendency of the right foot to drag slightly. On the 19th, whilst eating breakfast, his head had fallen forward on the table, and the right arm and leg seemed paralysed. Since this date the right foot dragged a little more and he would notice an occasional loss of control over the right arm and leg. On examination, arcus senilis was noticed; the pupils were small and responded well to light; tongue and face not paralysed; pulse 70, tense and regular; arteries rigid; muscular power of the hands equal, but in walking he drags the right foot slightly. On the 26th, the paralysis of the right lower extremity was more pronounced, and on the 28th paresis of the right arm set in. The discs were found, by Dr. J. L. Minor, to be the seat of double optic neuritis (of a lower grade on the right side). An internal purulent inflammation was diagnosed, and on May 1st, chloroform having been given, the skull was trephined. The dura mater was exposed, dark and nonpulsating. A hypodermic needle introduced withdrew dark-brown blood. The dura mater being opened the pulsations of the brain pressed fluid out which on examination consisted of brown-coloured serum and colourless red blood corpuscles. Four strands of disinfected horse-hair were used as drains. A perfect and uninterrupted recovery followed.

No. 23.—Drs. Bremer and Carson. *International Journal of Medical Sciences*. New Series. Vol. ciii., p. 134.—H. T. K., 21 years old, a healthy well-built youth, had become intoxicated, two weeks before being seen, and fallen between the joists of a new building. This he stated subsequent to his recovery. Save for a headache and occasional vomiting no evil results remained. A week later, whilst out walking, he suddenly became unconscious and fell. He was assisted home, and, undressing himself, went to bed. Dysphasia was found to be present, and the pulse slow and laborious. It was also noted that in the afternoon, when his temperature was 101° , he was more or less speechless, whereas in the morning when free from fever his dysphasia was less marked. He was first seen by Dr. Bremer thirteen days after the injury. The patient's condition then was quite rational, no injury to the head; motor aphasia of the ataxic variety was present combined with agraphia, paraphasia and paragraphia. Paresis almost amounting to paralysis of the right side of the face and tongue. All the limbs moved equally well. Sensation, muscular sense, and sense of temperature and pain were dulled on the right side. Next day, the grip of the right hand being weaker than the left, and the pulse having fallen to 43, Dr. Carson operated. Under the dura mater was found semi-liquid and clotted blood which extended as high as the longitudinal sinus above, and to the base below. A horse-hair drain was used. He was discharged May 18th.

No. 24.—Mr. Hulke. *Lancet*, 1883, vol. ii., p. 814.—The patient, æt. 60, an Irish day labourer, was admitted October 21st, 1881. He had had an injury fourteen days before and complained of darting pains passing through his head from a slightly ecchymosed lump in the right temple. His mind was quite clear. The accident was that a falling ladder, twenty-five feet long, struck him a glancing blow on the right temple, stunning him for a few moments. On the third day after the accident he had to give up work on account of severe headache. There was effusion of blood into the eyelids after the accident, but none into the conjunctivæ. On admission, he walked feebly into the ward, but no paralysis or loss of sensibility were present, the temperature was normal, the pulse 50, very compressible. On the fourth day in hospital he wandered in his mind. On the fifth day, paralysis of the right arm and leg occurred. His wife refused to allow him to be operated on. On the seventh day he was practically comatose with pulse rate of 70 and temperature 98.6° . On the eleventh day spastic rigidity of the left arm was first observed and this arm also occasionally twitched. An operation was now performed. The dura mater when exposed was healthy but bulged tensely and was non-pulsating. Three to four ounces of a brown flocculent fluid were evacuated. He made a steady recovery from the time of operation.

No. 25.—Mr. Butlin. *Medical Press and Circular*, vol. cxi., p. 357 (1895).—A man, 55 years of age, was hanging up paper on September 4th when he slipped and fell, striking his head against the corner of the mantelpiece. When admitted to the hospital he appeared badly shaken, there was bruising of the back part of the head in the middle-line. He had not lost consciousness. Next day there was some drowsiness and irritability. He remained like this until September 8th, when without any warning he was heard to cry out and the sister found him in a fit. He had seventeen fits before Mr. Butlin

arrived. In the fits he turned the head and eyes to the left, then the left upper limb became flexed, followed by the left leg and the extremities of the other side in that order. The rigidity was succeeded by clonic spasms which affected the left side of the face first, afterwards spreading to the limbs in the same order as the rigidity. He was conscious between the fits, but some paresis of the left side of the face and left upper extremity remained. Operation being decided upon, he was trephined at the junction of the middle and lower part of the fissure of Rolando. No sign of fracture was present, but beneath the dura mater was a clot no bigger than a finger-nail. This blood was not quite fresh, having evidently formed some days before. The arachnoid corresponding to the clot was somewhat raised from the pia mater by a collection of clear fluid. This serum was collected just over the fissure of Rolando, and it produced a depression of brain substance into which the finger could be put. It was drained with a strip of gutta-percha, the bone being put back. After the operation there were no more fits, but he was violent, excited and restless, complaining in his lucid intervals of headache. He required a male nurse until October 22nd, but ultimately recovered perfectly, as far as could be seen.

No. 26.—Mr. Cant. *Lancet*, 1891, vol. i., p. 542. Reported by Dr. Brock.—C. C., 34 years of age, a carter, was kicked on the right cheek and temple by a horse on September 25th. He was unconscious for some minutes after the injury, and then walked to the hospital. No fracture could be made out. He refused admission, so was dressed outside up to October 1st. On this day he had two fits for the first time in his life, and was admitted into the Lincoln County Hospital. On admission, he was perfectly conscious, and with no abnormal signs to be found. Urine 1015; no albumen present. He had eleven fits between the time of admission and 11 a.m. next morning. These fits began as a twitching of the left side of the mouth, followed by twichings of the hand and protrusion of the tongue to the right. During this time the right side and both legs were unaffected, but he now turned over on his back clenching his hands and drawing them up whilst the legs were extended stiffly, the eyes closed and the teeth clenched, the back being so arched that he rested on his head and his heels. This condition of opisthotonus lasted for a few seconds and then suddenly relaxed; at the same time he gave a loud cry and struck the bed violently several times, not rapidly but at intervals of five seconds drawing up his hands and arching his back, and then after a pause violently extending the arms whilst the back relaxed. The attack lasted four minutes. Immediately before he recovered he sighed deeply, turned on his right side, and put his right hand to his right temple, regaining consciousness perfectly in the course of a minute. The pupils were widely dilated, and he did not pass urine during the attack. Before a fit came on he had pain at the site of injury, which passed round his head, and he then lost consciousness. He continued having fits and getting weaker. The fits were longer and less violent, but the tongue was often bitten. On November 10th he was trephined over the right temporal region. No fracture of the skull was found, but on removing bone the dura mater bulged into the wound, and on opening it a quantity of yellowish fluid was found beneath the arachnoid and two small blood clots beneath the pia mater, which were removed. He was discharged December 22nd in perfect health and spirits, there having been no return of fits of any kind.

No 27.—Dr. I. G. Modlin. *British Medical Journal*, 1902, vol. ii., p. 704.—W. S., æt. 37, was admitted into the Monkwearmouth and Southwick Hospital on June 14th, with the following history. He had been drinking pretty heavily during the first week of the month. On the evening of June 9th he fell down, but no importance was attached to this fact. On the 10th he rested, and slept for the greater part of the day, on the 11th he went to work in a ship yard as usual, on the 12th he awoke in a feverish condition, with pains all over the body, was delirious, and with a temperature of 104°. He was prescribed for, and on the 13th was much better, the pain gone, and no delirium or fever present. At 2 p.m. on June 14th, fits set in suddenly. At first they were infrequent and slight, but as they increased in frequency and severity, he was sent to the hospital at 9.45 p.m. On admission, fits were recurring every two minutes. In the intervals between them the man was semi-conscious, and could answer questions. The face was flushed, the pupils dilated and fixed, the pulse slow and full. There was a slight indistinct bruise over the left parietal bone. The fits began as a blinking of the left eyelid, the muscles of the left side of the face were then affected, followed by the right side of the face. The movements then began in the fingers and toes of the left side, passing thence to the whole limbs. The right extremities were finally involved, though not as much as on the left side. It was decided to operate. On removing a crown of bone, a large clot was seen under the dura mater, no pulsation of the brain being evident. The membranes were incised, and clot found to extend round as far as could be felt. As much as possible was removed with the little finger and the scoop end of a director. A small drainage tube was employed. Another fit occurred a quarter of an hour after his return to bed, and nine more at gradually increasing intervals for the next five and a half hours, after which the man made an uninterrupted recovery.

No. 28.—Messrs. R. H. Cowan and E. H. Monks. *Lancet*, 1899, vol. ii., p. 1441.—A man of 55, was admitted to the Royal Albert Edward Infirmary, Wigan, on September 23rd, 1896. Four days previously he had fallen down stairs, and the day before admission had been seized with fits. On admission, he was conscious but aphasic, and his temperature was 100°. During the twelve hours following admission he had four attacks, each more severe than the one before. During the attacks the patient was unconscious, and the muscles of the right side of the face and right arm were in a condition of clonic spasm lasting several minutes. The only evidence of injury was a small bruise on the right side of the occiput, which was evidently superficial. He was trephined over the centres for the right arm and right side of the face. The dura mater bulged on removal of the bone. On incising the membrane a large blood clot presented. The clot was broken up, and washed away with boracic lotion. The wound was lightly packed with iodoform gauze and allowed to granulate up. Twitchings ceased entirely after operation. He spoke distinctly on the third day. He was discharged on November 7th, and in the February following he had been working as a collier for some weeks, and was quite well.

No. 29.—Dr. Charles L. Scudder. *Medical Record*, June 18th, 1898, p. 876.—M. A. B., a spinster, æt. 69, was brought to the Massachusetts General Hospital at about 10.30 p.m. She had fallen one and a half hours

previously, having been struck by a coasting sled. On examination, she did not know of the accident which had befallen her, or why she was at the hospital. She spoke coherently, but was suffering from slight shock. Pulse rate 64, respirations 16, temperature 97.5°. Bleeding from the right ear, and blood coming from the nostrils. No visible external injury, and no paralysis. The pupils were equal, contracted, and reacted to light. The patient was talkative, but not restless. During the night, however, she was restless, only sleeping two or three hours, and she vomited two or three times. At 3 p.m. the next afternoon the pupils were equal and reacting, she understood what was said to her, but did not speak coherently or distinctly. There was almost complete paralysis of the right arm, and paresis of the right leg. Pulse 85, full and bounding. No paralysis of face. At 4.30 p.m. the symptoms were considerably intensified, the breathing laboured and stertorous, and there were right hemiplegia and facial paralysis. Bleeding from the right ear continued to a slight extent all day. Middle meningeal hæmorrhage was diagnosed, and at 6.15 p.m. ether being given, the left middle meningeal artery was exposed. The blood was found within the dura mater, which was lacerated. The clot was temporo-parietal. The vessel was tied, and a drain, reaching to the dura, inserted. The temperature rose to 110° F. during the night, but fell immediately, and gradually came down to normal. The paralysis began to clear up on the 6th day from the leg, and the 10th day from the arm. The 30th day she walked alone, and left the hospital.

No. 30.—R. Lépine. *Revue de Médecin*, vol. xvi., 1896.—A man, 29 years of age, very intelligent, but an incorrigible drunkard, and subject to epileptic fits, fell downstairs on June 21st, 1889, and was rendered unconscious. Two days later he was brought up to the Hotel de Dieu (de Lyon). On admission coma was absolute, but no signs of external injury were present, and no discharge from the ears or the nose. In two days the coma gradually disappeared, but aphasia remained. In addition, slight paralysis of the face and right extremities was present, whilst the tongue deviated to the right. After this, epileptic fits, Jacksonian in type set in, always starting with twitching of the right labial commissure, immediately followed by convulsions affecting the right extremities. Ten days after the fall, as there was no improvement, Dr. Jaboulay trephined over the lower part of the left Rolandic area. At the moment that the dura mater was incised, there spouted out about twenty-five grammes of a brownish red liquid. Antiseptic dressings were applied. After this the aphasia and paralysis gradually cleared up. The patient left the hospital in December. Later he had some slight attacks of epilepsy, but these have not prevented him from working in a dyeing manufactory, and they have not recurred.

No. 31.—Dr. Hughlings Jackson. *London Hospital Lectures and Reports*, vol. iv., 1867, p. 352.—Mrs. S., æt. 80. On Saturday, February 9th, she was standing on the top of a short flight of stairs when she felt stupid and remembered nothing further until she found herself at the bottom of the stairs. On being picked up she said she was not much hurt, and went up another flight of stairs, being seen by another neighbour who thought she looked dazed. She lay on her bed, but later undressed and went to bed. On February 10th she seemed comfortable, and spoke well. On the 11th she

was found on the floor insensible, having, it was supposed, got out of bed to make water. She was seen this day by Dr. Hughlings Jackson with Dr. Roberts. She was still insensible, and for four hours (ending 8.30 a.m.) had had a series of fits. In each fit the right side of the face had been affected. It was said that both arms were affected. At 10.30 a.m., the hour she was examined, she took no notice of what was said to her. Occasionally she opens her eyes, and sometimes moves her left hand to her head. Continually moans. Once only she moved her right hand for a very little distance. She drew up both her legs and kept them up. Eyes were closed and there was a deviation to the left. The pupils were unequal and small, but not abnormally so. Pulse 96. Respiration 36, with each expiration was a moan. On the 13th she was still insensible. Pulse 100. Respiration 34; no stertor. The right arm was apparently paralysed. Her mouth was constantly moving and she moaned. During the night she had had constant fits. In these the working of the mouth increased, the right cheek and right orbicularis palpebrarum twitched, the jaws moved together, the head and eyes turned to the right a trifle more than in the interval, the respiration ceased, but the thorax and abdomen seemed to jerk a little. When the fit ended several deep breaths were taken. The arm in most attacks was unaffected, but in two others there was a very little jerky movement in it. February 14th, she had been in fits nearly the whole night, the right arm being now involved but the leg never moved. During the evening the respirations were 48 per minute and were very shallow, she had fewer fits and died. She only spoke four times during this period of unconsciousness. Three times she called a daughter's name (Isabella) and once replied "Yes" to a question (relatives' testimony). At the post-mortem: There was much blood under the dura mater, on the left side only, from front to back of the hemisphere and at the base. The bulk of it was pressing on the frontal convolutions. The blood was above the arachnoid. About half-way up the fissure of Rolando was an opening in the arachnoid blocked by a piece of clot, and through this it was believed the blood had come. The heart was soft and small, the valves competent, but the aortic ones very atheromatous. The kidneys were healthy.

No. 32.—Dr. Harrington Sainsbury. *Pathological Society's Transactions*, vol. xxxviii.—J. M., a woman, 42 years of age, was admitted to the Royal Free Hospital under Dr. Samuel West on December 1st, 1886. On admission, she was in a semi-comatose condition which persisted more or less till death on January 2nd, 1887. Obscure paralytic symptoms were present, referable chiefly to the right side. The woman was a drinker and had recently drunk heavily. She was reported to have fallen four days before admission. There was bruising over the right hip-bone. No albumen in the urine. At the autopsy, there was blood-clot covering the fronto-parietal region of the right hemisphere and extending into the anterior and middle fossæ of the right side. The dura mater was rather firmly adherent to it in some places. The clot was placenta-shaped. No source of hæmorrhage was discovered. The dura mater over the left side of the brain was perfectly healthy.

No. 33.—Dr. D. G. Denton. *Lancet*, 1898, vol. i., p. 690 (Report of Meeting of Æsculapian Society).—An alcoholic man received a blow two inches above the left ear. He was picked up at once unconscious. On the second day

with return to consciousness headache was complained of. On the eighth day the pulse-rate was 60, the temperature 97°. He died suddenly on the ninth day. At the autopsy a blood-clot weighing one and a half ounces was found adherent to the brain. The viscera were not healthy.

No. 34.—Dr. Molleson. *Journal de Médecin*, Août, 1766, Paris (reported by Guthrie in "Injuries of the Head," p. 65).—A young man received a blow on the right parietal bone, which wounded the integuments without causing fracture. Coma, with paralysis, followed, and he bled from ears, eyes, nose and mouth. Trephined over the right parietal bone, dura mater opened, and blood escaped. The patient was relieved. Five days later he complained of pain on the left parietal bone, which was soon followed by fever and delirium. Bone exposed on this side and a fissured fracture found. Trephined, and dura mater opened, from under which a glassful of dark red fœtid matter was evacuated. The patient recovered.

No. 35.—Dr. Petit (from Guthrie's "Injuries of the Head," p. 125).—A grenadier, struck on the side of the head by the splinter of a shell was knocked down on the glacis, but on being carried away soon recovered his senses. He had scarcely arrived at the hospital when he fell into a state of stupefaction. He was again bled, and a tumour which had formed over the temporal muscle was laid open. The bone beneath was not fractured, neither was the periosteum detached. As the symptoms of pressure persisted the trepan was applied, but no extravasation was discovered underneath. Five or six hours after the operation he spoke and answered some questions, took some nourishment, but relapsed shortly afterwards into a similar state of stupefaction. On removing the first dressing, the cause of evil was made manifest. The dura mater had risen into the opening made by the trepan, and was above the level of the bone, which had given some relief to the compressed parts, and had probably been the cause of the temporary amelioration which had taken place. The dura mater was opened by a crucial incision, and two table-spoonfuls of half fluid, half coagulated blood were evacuated. His stupefaction ceased two hours afterwards, and he recovered so as to be removed with the other wounded who were nearly cured. (Originally reported by Dr. J. L. Petit in his work "*Des plaies de la Tête dans le Traité des Maladies Chirurgicales*," Paris, 1730).

No. 36.—Dr. (now Sir) William McEwen. *British Medical Journal*, 1888, vol. ii., p. 305.—A boy had fallen six days previously, causing some slight bruising about the face and head, and accompanied by a shade of mental obscurcation. Forty-eight hours after the injury he appeared so well that his parents wanted to let him get out of bed. On the sixth day, however, a series of convulsions seized him. Twitchings began in the left side of the face and gradually involved the left arm, and subsequently the left leg, during which consciousness was preserved. Paresis of these parts remained, though sensation was unimpaired. On the following day the convulsions persisted, finally becoming general, with loss of consciousness. When the head was shaved for operation, a scarcely perceptible irregularity was detected in the cranial vault near the coronal suture. The skull was exposed, with a fissure running across the coronal suture. Trephined. The dura mater had a dark colour, and on opening gave vent to two ounces of fluid and coagulated blood contained in the subdural cavity. Patient made an uninterrupted, typical afebrile recovery.

CLASS B.

No. 37.—Mr. Golding-Bird. Guy's Hospital Surgical Reports, 1902. No. 74.—A. S. W., 32 years of age, fell out of a train and was admitted into Guy's Hospital on January 24th with a compound depressed fracture of the skull. He was quite conscious on admission, but half an hour later became drowsy, and in four hours was unconscious, with stertorous breathing. Pulse 94. Respirations 42. He was trephined at the seat of fracture and the bone elevated. Extradurally there were two ounces of dark blood. As the dura bulged, a small hole was made into it; cerebro-spinal fluid spurted out to a height of six inches. At first clear, it was later mixed with blood. The patient's conjunctivæ were blood shot and dark, and it was thought that the base of the skull was fractured. Death occurred nine hours after the operation, and thirteen hours after the injury. No post-mortem record is to be obtained.

No. 38. — Sir Henry Howse. Guy's Hospital Surgical Reports, 1901. No. 522.—A. G. H., a waterside labourer, 30 years old, was knocked down by a heavy van in the Old Kent Road. On admission, there were two scalp wounds, both extending to bone. He was unconscious and remained so till death. There was no bleeding from the nose, ears or mouth, but he vomited blood. Pulse 42, feeble. Respiration 28, stertorous. Temperature 95°. The pupils were normal and reacted to light, but afterwards alternately contracted and dilated. Later still, the right eye became widely dilated and remained so, whilst the left eye had an internal strabismus. Arms and legs rigid. No involuntary passage of urine or fæces. He was trephined some hours after admission, first on the left side where no blood was found, afterwards on the right side where subdurally blood was found. He died five hours after operation, the temperature rising to 107° before death. At the post-mortem examination a transverse fracture of the occipital bone with laceration of the brain was found.

No. 39.—Mr. Golding-Bird. Guy's Hospital Surgical Reports, 1901. No. 195.—A. H., æt. 50, admitted March 24th, 1901. An iron plate had fallen on his head whilst at work. He walked home and went to bed. Some hours later he fell into a comatose condition, and was brought up to the hospital on an ambulance. On admission, his pulse-rate was 98, and in character it was full and easily compressible. The temperature was 99·4°, but higher in the right axilla than in the left. Respiration 30 to 35, somewhat forcible. In a semi-conscious condition with an irregular, bleeding scalp wound in the occipital region, not reaching to bone. He was able to move his limbs, but there was considerable rigidity in the limbs of both sides, more in the left than the right. Both knees were stiffly semi-flexed, and observed to twitch once or twice. Left pupil larger than right. On the 25th, he was much the same. On the 26th, he was worse, and at 1.30 p.m. spasmodic twitchings of the face, almost entirely confined to the right side, and of the right hand, set in. These attacks recurred every five to fifteen minutes. At 6 p.m. Cheyne-Stokes' respiration set in. He died at 1.30 a.m. on the 27th. At the autopsy the whole of the left side of the cerebrum was covered with subdural hæmorrhage. The anterior pole of the frontal lobe was severely lacerated.

Small amount of subdural hæmorrhage on the right side also. On cutting open the brain a large blood-clot was found in the anterior horn of the lateral ventricle (left), which communicated through the lacerated anterior frontal lobe with the subdural hæmorrhage. No fracture of the skull. No indication of arterial disease at base of the brain. Kidneys, heart, liver and spleen normal.

No. 40. — Sir Henry Howse. Guy's Hospital Surgical Reports, 1900. No. 171.—G. H. D., a labourer, æt. 19, was struck on the head by a passing express train, and picked up unconscious. He was brought up to the hospital from Norwood Junction. On admission, his pulse rate was 58, his temperature 97·8°. He was unconscious and bleeding from wounds in the right temporal region which exposed bone. Bleeding from the nose and hæmatemesis. Pupils equal and moderately dilated, reacting to light on the left side, but very slightly on the right. Knee-jerks exaggerated in both limbs. Later twitchings set in in the fingers, then the arms and legs, and finally over the whole body. Decided to trephine. Right temporal region exposed, found fractured. A large clot was found between the dura mater and the skull. This was removed. The dura mater was then punctured, letting out a large quantity of dark coloured blood which kept welling out of a ruptured cerebral vein, which was therefore tied. The brain was pulped in this region. The patient died the same evening at 9 p.m. without recovering consciousness. No post-mortem examination.

No. 41.—Mr. Golding-Bird. Guy's Hospital Surgical Reports, 1900. No. 621.—Joseph D., æt. 36, an occasional drinker, fell downstairs on December 23rd, and was thought to have injured his head. He was put to bed, no symptoms being noticed by his friends. On the 24th, curious movements of the eyeballs were noticed by his landlord, and that he was not in complete possession of his faculties. He was brought up to the hospital by two men, being able to walk with their assistance. He was told to come up each day if he should not get well. On the evening of the 24th and the morning of the 25th, he suffered from delusions. On the 26th and 27th, he was brought to the hospital, and was thought by his friends to be more delirious. On the 28th, bleeding from one ear was noticed, and towards midnight he began to have fits which recurred at intervals of a quarter to half an hour. These fits started in the left hand, spread to the limb, the left side, and finally involved the right side. He was admitted to the hospital at 8 a.m. on the 29th. On admission, pulse 136. Temperature 101·8°. Respirations 92, stertorous. Quite unconscious. A strong, muscular, well nourished man. Incontinence of urine and fæces. Nystagmus in both eyes. Fits at intervals of a quarter to half an hour. There was sweating, and the temperature was higher in the left axilla than in the right. Locally there was bruising in the right parietal region, and some dried up blood in the external auditory meatus. The fits becoming more violent, operation was decided upon, and at 11.30 a.m. a flap was turned down in the right temporal region. A fracture along a line passing backwards over the right ear was found. A large amount of partially dried up blood clot was present above the dura mater. On its removal blood welled up from below, and was thought to come from the lateral sinus. Fluid was thought to be under the dura mater, and on incising this membrane fluid

came out under a high pressure. A small incision was made into the pia mater, and the brain substance found to be infiltrated with blood. During the rest of the day there was no return to consciousness. On the 30th, there were signs of returning consciousness in the early morning, but at 9 a.m. fits set in again much as before, and although the wound was opened up and the subdural space drained the fits persisted, and he died at 5.30 p.m. At the autopsy, fracture of the base on the right side was found, with ecchymoses over the whole surface of the brain, and extensive subdural hæmorrhage over the right temporo-sphenoidal lobe.

No. 42.—Sir Henry Howse. Guy's Hospital Surgical Reports, 1899. No. 112.—James R., æt. 45, a brewers' carman; was admitted at 8 a.m. on the 2nd of October, 1899, shortly after falling from his dray on to his head. He was unconscious for a quarter of an hour after the accident. On admission, he was dazed, but not alcoholic, the pupils were equal and dilated and there were no paralyses. There was a severe bruise at the top of the head with the skin slightly broken; no bleeding from ear, nose, etc. Seemed rather better when admitted into the ward, and helped to take off his clothes. Pulse 80. Temperature 97.8°. During the night he was quiet except for one occasion when he tried to get out of bed. At 8 a.m. next morning he became comatose, with stertorous breathing. Pulse 120. Temperature 102.6°. Mr. Jacobson saw the patient and decided to operate. The skull was trephined. The dura mater was first punctured with a needle, when clear serum came away. Later blood clot was removed subdurally. The patient never came round from the coma, and died about an hour after the completion of the operation. The temperature before death was 104.8°. At the autopsy, there was extensive subdural clot along the left side of the brain, the left frontal lobe was lacerated at the base, and the base of the skull was fractured on the left side over the bend of the lateral sinus.

No. 43.—Mr. Lucas. Guy's Hospital Surgical Reports, 1889. No. 274.—Edwin G., æt. 31, fell downstairs, and was picked up unconscious. On admission to the hospital he could be roused, but was occasionally violent. Both pupils were equal and contracted, and reacted normally. No signs of fractured base no paralysis or rigidity. Pulse 50, full. Temperature 97°. There was swelling across the back of the head and over the right eye. Became suddenly comatose, the pulse went up to 72, the left pupil became larger than the right and did not react to light, the limbs were all flaccid and the breathing stertorous. Operation being decided upon, a flap was turned down in the left temporal region. A fissured fracture of the skull was found. The skull was trephined and the dura mater exposed, tense and bulging. On incising it, a lot of clot was found and removed. The brain below was seen to be lacerated, and the whole vault of the cranium appeared to be loose. The patient died four hours after the operation. At the post-mortem examination a longitudinal fracture through the base with rupture of the lateral sinus was found. Subdural blood clot with laceration of the temporo-sphenoidal lobe.

No. 44.—Mr. Lucas. Guy's Hospital Surgical Reports, 1899. No. 705.—William B., æt. 43, was admitted at 6.30 p.m., on December 1st, 1899. He had fallen 15 feet head downwards, and a heavy door had fallen on him. He was

picked up unconscious. On admission, he was unconscious with stertorous breathing. Blood came profusely from both nostrils, especially the right. The left arm was twitching, the left leg rigid. The pupils were contracted to the size of pin-points. There was a compound depressed fracture affecting the right parietal bone. The patient was taken to the theatre, and A.C.E., followed by ether, given. Extensive fracturing of the skull was found, and extradural blood clot was removed in quantity. The dura mater was then found tense, barely pulsating, and dark in colour. Beneath it a large clot was found compressing the Rolandic area. The clot was removed, and hæmorrhage controlled by a plug of gauze low down in the subdural space. The dressings repeatedly became soaked through and had to be changed. The patient was infused just before he died. No post-mortem examination was made.

No. 45.—Mr. Golding-Bird. Guy's Hospital Surgical Reports, 1899. No. 427.—Phoebe G., æt. 59, a heavy drinker, fell a distance of 16 feet from a window to the pavement. Her head struck the ground first. She was picked up unconscious and bleeding from the left ear. Two hours later she was brought up to the hospital, having vomited twice on the way up. On admission, she was unconscious, with a large bruise half-way down and just anterior to the left parieto-occipital suture. Pulse 72, soft, irregular. Respirations 28, stertorous. The right pupil larger than the left, both reacting to light. She regained consciousness in an hour, becoming irritable and restless, and soon falling into a state of drowsiness. At 10 p.m., when it was decided to operate, she was unconscious, with very stertorous breathing, and the pupils were uneven, the left being widely dilated, and neither of them reacting to light. The skull was trephined over the region of the left middle meningeal artery. The dura mater was found tense and non-pulsating. The membrane was incised, when some serum and then a quantity of blood clot were removed. Drainage with gauze was used. The patient was obviously relieved by the operation, but the coma persisted, and she died four hours after the operation. No post-mortem examination was allowed by the coroner.

No. 46.—Mr. Davies-Colley. Guy's Hospital Surgical Reports, 1899. No. 70.—Mary L., æt. 64, fell downstairs and struck her head. She was unconscious when picked up, but when a friend returned with a doctor she was conscious again and had vomited. She was sent up to the hospital and admitted on the 22nd January. On admission, she was conscious and answered questions well. There was bleeding from the mouth and left ear, and she vomited blood. Pupils equal, breathing easy. The pulse was 96 and rather irregular. The right cheek was puffing slightly with expiration, otherwise there was no paralysis or rigidity. Next day she was much the same. On the 24th the patient was unconscious, there was a bruise immediately in front of the mastoid process, and extending upwards towards the temporal ridge. On pressing this bruise the left arm moved, the right arm remaining still. Twitching of the left side of the face was marked. The right side of the face was apparently paralysed save for the corrugator supercillii muscle. The right eye was fixed, insensitive, pupil contracted but reacted very slightly to light. The right eye was quite sensitive and reacted to light, but the pupil was contracted. Legs both drawn up on pressure on the bruise on

the head. On the 25th, there was no change, whilst towards evening fits set in, confined for the most part to the right side of the body and head. These started in the chin and spread in order to face, arm and leg. The eyes were directed on each occasion to the side on which the fit occurred. She was trephined over the left lower Rolandic area. Extradural hæmorrhage was found. The dura mater was opened, and a large quantity of blood and serum escaped. The brain was much depressed, but pulsating. The blood was thought to come from the middle meningeal artery, but pressure on the artery did not arrest the bleeding. The hæmorrhage was stopped with pads, the dura mater stitched with catgut, the trephine bone replaced, and the wound sutured. Next day the patient was much better, she could use the right arm freely, and the facial paralysis was less marked. Her condition was quite satisfactory up to February 10th, when she became irritable and refused food. On the 12th the wound was opened, the dura mater incised, and a large abscess found in the left hemisphere. She died next day.

No. 47.—Mr. Davies-Colley. Guy's Hospital Surgical Reports, 1898. No. 427.—Dave S., 55 years of age, was brought to the hospital on September 5th with a bruise and small scalp wound at the back of the head. He was very drunk, and the cause of his injury was never discovered. He was admitted to the surgery at 1.30 p.m. and detained till 5 p.m. He several times tried to escape, and finally before being discharged in charge of the police at 5 p.m., he was washed out. At 10.30 p.m. the police brought him back and he was admitted to Cornelius ward. At this time he was quite comatose, with the right arm and leg paralysed. Pupils both dilated and not reacting to light. Pulse 80, full and strong. Respiration stertorous. No facial paralysis. An hour after admission he was operated upon. Chloroform was given, and the left Rolandic area exposed. The skull was very thick, and while removing the piece of bone with the elevator it broke across quite easily near the centre. Very little force was used, and it was considered that there must have been a pre-existing fracture there. The line of fracture was nearly parallel to the zygoma. No extradural hæmorrhage was present. On opening the dura mater a large blood clot was exposed to view and removed. The clot had a diameter of four inches, and its removal was followed by severe hæmorrhage. The wound was temporarily packed, and then irrigated with warm boracic lotion. The wound in the dura mater was sewn up, the bone replaced in pieces, the skin sewn up, a gauze drain being inserted. Next day he was much better, and the improvement continued until 4.30 p.m. on the 7th, when he suddenly became worse. Improvement again followed, but he ultimately died at 11.25 p.m. on the 8th. At the autopsy a large clot was found between the dura mater and the brain, whilst the brain itself was extensively bruised. There was no fracture of the skull, no laceration of the lateral sinus, and no sign of rupture of middle meningeal artery.

No. 48.—Mr. Durham. Guy's Hospital Surgical Reports, 1894. No. 3.—Edward B., æt. 42, lighterman, was admitted into Cornelius ward on December 20th, 1893. He fell backwards whilst going upstairs in an alcoholic condition. On admission, he had stertorous breathing, pupils contracted and insensitive to light, pulse regular, urine 1,020, no albumen. There was a hæmatoma over the parietal and occipital bones of the right side. The

muscular resistance was fairly good when the arms were raised. On the 21st he was drowsy, but could be roused. For the next week he was semi-conscious, restless, and at times noisy. On the 28th he was moved up to the strong room (pulse 74), and apparently passed through an attack of delirium tremens. On the 31st he was somnolent, but could be awakened (temperature 97°, pulse 74). On January 1st he was comatose. At 11 a.m. the pupils were equal, as also the tone of the muscles on the two sides. At 2 p.m. the left pupil was larger than the right, there was slight facial paresis on the left side, breathing stertorous. Pulse 58. Operation was performed at 2.30 p.m. The dura mater was found bulging and not pulsating. On incising it, there came a gush of dark-brownish blood. About two or three ounces of blood came away altogether. The opening in the dura mater was left open for drainage, but no tube used. There was some improvement after operation, but the coma persisted, and the patient died shortly after 12 a.m. on the 3rd. At the autopsy blood was found covering the right and left frontal lobes beneath the dura mater, the poles of the frontal and temporo-sphenoidal lobes were contused more on the left side than the right. There was a fracture of the posterior fossa. The left kidney was small and puckered, the right large, with one scar.

No. 49.—Sir Henry Howse. Guy's Hospital Surgical Reports, 1891. No. 289.—John M., æt. 36, fell from the side of a dock into a moored barge, a distance of about twenty feet, and was admitted to Guy's at 11.30 p.m. on January 28th. On admission he was unconscious, wholly paralysed, bleeding from both ears and both nares; respirations slow and shallow; pulse about 50, feeble. At 2 a.m. on the 29th he recovered the use of the left arm. Trephined at 9.30 a.m. in the left temporal region. The brain was found congested, but no clot present. He died two hours later, with stertorous respiration and pupils dilated and fixed. At the post-mortem examination, there was a large effusion into the right temporal muscle. There was a large temporo-parietal subdural clot half an inch thick on the right side. The blood came from the vessels of the pia mater. The brain was severely bruised, and there were severe fractures of the skull.

No. 50.—Mr. Durham. Guy's Hospital Surgical Reports, 1890. No. 297.—Walter B., æt. 28, fell from the dickey of his van on to his head on July 10th and was brought up to the hospital. On admission he was excitable. There was a scalp wound over the lambdoid suture exposing periosteum. The pupils were equal and reacted. No paralysis. No signs of fractured base. He was alcoholic, and soon after admission was removed to the strong-room on account of his noisy behaviour. He was brought back to the ward on the 11th, at 2 p.m. On the 13th he was quiet and knew his friends. (His mother stated that he was a heavy drinker and had had a severe blow on his head before.) On the 14th he had fits. He had had three slight ones involving the left side of the face during the night. At 9 a.m. he had one which spread to the left arm and leg and then the right arm and leg. He remained unconscious, with stertorous breathing between the attacks. The fits recurred at intervals of one and a half to two minutes. On the 15th the fits were less. He had had two during the night but less violent than before. Temperature 98.6. On the 16th, he was unconscious, with stertorous breathing. Mr. Lane

examined the eyes with the ophthalmoscope and found double optic neuritis. He remained the same till 7 a.m. on the 17th, when he seemed to have a choking fit and died suddenly. At the autopsy there was a V-shaped fracture, with limbs horizontal, in the right occipito-temporal region. There was blood both internal and external to the dura mater. The former amounted to one and a half ounces. There was extensive pulping of the orbital convolution, and the pole of the temporo-sphenoidal lobe on the right side. The left side of the brain was normal. The heart was healthy, the kidneys large and tough but otherwise normal; the liver weighed seventy-three ounces.

No. 51.—Mr. Durham. Guy's Hospital Surgical Reports, 1890. No. 326. —Henry T., æt. 32, was knocked from the seat of his van to the road, the result of a collision with an omnibus. On admission (October 3rd), he was irritable, would not keep quiet, and seemed under the influence of drink. He vomited a lot. There was bleeding from the left ear, but no scalp wound or hæmatoma. He was noisy, and tried to get out of bed the following night, necessitating his being strapped down. Temperature 101·2°. No albuminuria. On the 5th he was transferred to the strong-room. On the 6th he had a fit at 6.30 p.m., and had twenty-three up to 8 a.m. on the 7th. The fits start with deviation of the head and eyes to the left, then twichings began in the left eyelid and spread to the whole of the left side not affecting the right. During the fit both pupils contracted. Between the fits he was conscious and had left hemiplegia. Temperature 100·4°. He was given chloroform and trephined in the right temporal region. The dura mater was exposed tense, and on incising fluid squirted out four inches high. Blood clot was present and removed. On the 9th he was conscious: the hemiplegia had cleared up, but the facial paralysis on the left side persisted. On the 10th he appeared well, answering questions and speaking intelligently. On the 11th bad symptoms set in, and on the 12th he died, the temperature reaching 104° before death. At the post-mortem examination there was diffuse meningitis. There was bruising of the right temporo-sphenoidal lobe. There were fractures of the middle and posterior fossæ of the skull.

No. 52.—Sir Henry Howse. Guy's Hospital Surgical Reports, 1890. No. 276.—Alfred N., æt. 60, was knocked down by a cab on November 7th. He was able to walk into the surgery at about 6 p.m. The pulse was rapid, and he had a wound at the back of the head, not, however, exposing the bone. At 8 p.m. his breathing had become stertorous, the right pupil was dilated and fixed, the left being normal; all the limbs were rigid: the pulse was 51 and full. He was trephined over the right middle meningeal artery. A fissured fracture of the skull was found and through it most of the blood from a rupture of the middle meningeal artery had escaped into the tissues of the scalp. The dura mater being opened, the front part of the brain was found lacerated, and there was a temporo-parietal clot. Next day he answered when spoken to; the right pupil was a shade larger than the left, and both react to light; there were no unilateral symptoms. On the 9th he seemed to be progressing favourably, but on the 10th, at 10.30 a.m., the temperature was 104·6°; at 2.15 the respirations were stertorous (48), the pulse 160, and the temperature 104·2°. The optic discs were completely blurred over. He died at 3.30 in the afternoon. At the post-mortem,

the fracture found at operation extended to the occipital bone, but there was no fracture of the base. About one ounce of blood was found internal to the dura mater, and a small quantity of blood superficial to the dura mater. No meningitis. The right temporo-sphenoidal lobe was lacerated and soft. No atheroma of vessels; kidneys normal and heart fatty.

No. 53.—Mr. Davies-Colley. Guy's Hospital Surgical Reports, 1888.—John B., æt. 35, fell downstairs on to his head, and was admitted into Guy's Hospital at 11 p.m., on March 11th, 1888. On admission, he was comatose, with stertorous breathing. The pupils were unequal, the left being contracted. Over the right parietal bone at its posterior part was a large bruise. At 11.30 p.m. Cheyne-Stokes breathing was present; the pulse was 40, labouring, the respirations 38; the left pupil dilated. At 12 midnight the left pupil had contracted again. On the morning of the 12th the coma still persisted; the respirations were 30 per minute, sometimes almost Cheyne-Stokes like in character and sometimes stertorous; pulse 60, rather small; left pupil contracted. When seen by Mr. Davies-Colley at 1.45 p.m. the left pupil was more dilated, but still a good deal smaller than the right. Marked Cheyne-Stokes respiration was present. Trephined over fore part of right temporal region. On opening the dura mater some dark fluid blood at first came out, then black clot. Mr. Davies-Colley passed a finger between the dura mater and the brain till it rested on the dura mater covering the lesser wing of the sphenoid. On withdrawing the finger, more clot came out and some broken-down brain matter. The patient improved for a time after the operation, but died with Cheyne-Stokes respiration and pulse-rate of 40 at 2 a.m. on the 13th. At the post-mortem examination there was an extensive fissured fracture of the vertex, and a small localised fracture of the left middle fossa at the base. The fore part of the right frontal lobe was pulped. The viscera were healthy.

No. 54.—Sir Henry Howse. Guy's Hospital Surgical Reports, 1887. No. 224.—Henry Brown, æt. 25, was thrown from his waggon on to his head on February 5th. He walked home and became drowsy. On February 7th he was admitted to Guy's Hospital in an unconscious condition (11.15 p.m.). On admission, he became restless when irritated, but did not speak. His movements are confined to the left side of the body. Pulse 80, irregular; pupils equal, active and moderately dilated; right facial paresis. No bruising of head found and no signs of fractured base. At 3.30 a.m. on the 8th he had a fit beginning with vertical oscillation of the eyes and movements of the left arm. These increased, spreading to the mouth and face and then to the legs. The fits continued during the day and the next night became increasingly frequent and involved the whole body as clonic movements. At 10 a.m. on the 9th he was quiet, but now had paralysis of the left arm and paresis of the left leg. The left eye became inflamed, apparently from exposure, owing to the orbicularis palpebrarum not acting. At 3.30 he spoke, asking for a drink, which he took. On the 10th he seemed better, the left hemiplegia had disappeared, but he was restless and tried to get out of bed. On the 12th he had recovered his senses largely, attempting to protrude his tongue and whistle when asked to. Pupils equal; internal strabismus of left eye. Pulse feeble. Temperature 98.4°. Can move his arm and legs perfectly. After this he became restless again and relapsed into

a condition of coma, finally dying at 9.15 p.m. on February 16th. At the autopsy, about 3 ounces of clot and brown-coloured blood were removed from beneath the dura mater on the right side. The clot was temporo-parietal chiefly, but extended forwards somewhat. The general surface of the brain was uninjured, but there was an area about one inch in diameter over the second and third right temporal convolutions where the arachnoid and pia mater enclosed a hæmorrhage, and with a depression due to destruction of brain substance which was traversed by a fair-sized vein, which was probably lacerated and the origin of the blood. The brain was quite healthy, and the post-mortem report (No 45, 1887) says, "It appears probable that had the clot been removed he would have recovered." Viscera healthy.

No. 55.—Mr. Bryant. Guy's Hospital Surgical Reports, 1884. No. 279.—Thomas C., æt. 56, was kicked in the abdomen by a horse, and, falling on some stones, struck the right side of his head. He was rendered unconscious, and was still in the same condition when admitted into Guy's Hospital at 1.40 a.m. on September 6th. A wound was present in the occipito-parietal region which did not expose bone. The pulse was slow, regular, the breathing stertorous, and the pupils contracted, the left more so than the right. Both arms were rigid, the legs slightly so. At 10.15 there was paresis of the right side of the face, right hemiplegia and divergent strabismus. On the 7th he remained much the same, but the breathing was quieter. On the 8th he was noisy, recognised his wife and asked for a drink. On the 9th he was becoming cyanosed. Pulse-rate 90. Respirations 60. He died on the morning of the 10th, the right hemiplegia remaining till the end. At the autopsy there was a fracture of the right side of the skull, bifurcating in the middle fossa. Subdural hæmorrhage over left side of brain with superficial laceration. The heart was normal, the vessels good, the kidneys granular. Liver very tough. Testicles healthy.

No. 56.—Mr. Durham. Guy's Hospital Surgical Reports, 1878. No. 324.—William G., æt. 25, was thrown out of a cart and pitched on his head two days before admission. He was picked up insensible and carried home. After some time he regained partial consciousness and was admitted into hospital like this on January 9th. It was said that he had been very sick and vomited up some blood. On admission, he was semi-conscious and could scarcely be roused, complained of headache, and had both pupils dilated, the right more than the left. There was slight paresis on the left side of the body, but no facial paralysis. Small scalp wound in the occipital region. On the 10th he remained much the same. Temperature 98.4°. Respirations 16. Pulse 56. On the 11th he appeared to be better, putting his tongue out and moving his arms and legs when requested. Slight facial paralysis on the left side. At 9 p.m. he had two fits and another at 9.20. After this last one he remained unconscious. The fits resembled epileptic fits, the movements beginning on the right side. The fits continued to increase in frequency, and he died suddenly after the fortieth. At the post-mortem examination, a thin layer of blood clot covered the anterior part of the left hemisphere and frontal lobe of the right side. The brain was bruised extensively but superficially on the under surface of the middle and anterior lobes. There was a fracture of the middle fossa.

No. 57.—Mr. H. W. Allingham. *Clinical Society's Transactions*, vol. xxii. —Nathaniel C., æt. 40, admitted into the Great Northern Hospital, December 7th, 1888. He had fallen in getting off a tram-car, but did not remember the circumstance himself. Found to be somewhat concussed and complained of pain in the left shoulder, but nothing objective was to be seen there. No external signs of injury to the head were found. Given a draught of chloral and bromide and slept well. Next day he was rather drowsy, with pains in the right side of the head. The pupils were equal and reacted to light. December 11th, remained in the same drowsy state; irritable when disturbed or examined; at times rather light headed. Highest temperature yet 100.6° on December 9th. No paralysis; pupils normal; no vomiting; no albuminuria. On the 13th, at 6 a.m., convulsions set in. They began in the muscles of the left side of the face and affected the left limbs. Right pupil larger than left. Stertorous breathing. Fits recurred at frequent intervals. Mr. Allingham decided to operate. Posterior branch of middle meningeal artery exposed. A dark mass appeared beneath the dura mater, which did not pulsate; it proved to be a large black clot of blood which was removed leaving a large cavity extending almost as far as the finger could reach. This was irrigated with carbolic lotion till all clot had been washed away. The brain seemed a good deal lacerated over the right frontal lobe. Drainage tubes, two in number, were inserted. The man made a good recovery, and there is an additional note that on July 1st he was quite well and suffering no inconvenience at all.

No. 58.—Mr. Cock. (Reported by Sir Samuel Wilks in vol. v. *Guy's Hospital Reports*.)—A. W., æt. 25, was thrown down in a scuffle and struck his head on the pavement on October 21st. He was picked up insensible, taken to a neighbouring hospital, where his wound was dressed. He was then sent home. On the 22nd he was in a half-stupid state and had what was thought to be an epileptic fit. Another of these fits occurred on the 23rd. On the 24th he was sent to Guy's Hospital, where he appeared to be in a state resembling concussion and with a scalp wound on the left side behind. In the evening he was restless, with delirium. On the 25th he was quiet all day. That night, however, he had another epileptic fit which left him in an almost maniacal condition, from which he sank into a half-comatose state, occasionally screaming out. The pupils became contracted at the last, and the sphincters paralysed. He died November 15th. At the autopsy, there was no injury to the bone. On the right side there was a subdural collection of blood one inch thick. It existed more on the anterior and lateral parts towards the base. The clot was dull red in colour, and in some parts yellow, showing age. At two portions of the base yellow clot was distinctly adherent to the brain, and on removing it the latter was found to be bruised and soft. "Thus it is tolerably clear that the hæmorrhage was from some ruptured vein of the pia mater at this spot." None of the large arteries or venous sinuses were found injured. No inflammatory products were found in any part of the brain.

No. 59.—Mr. Jonathan Hutchinson, Senr. *Clinical Lectures and Reports* (London Hospital), vol. iv., p. 10.—A sailor, 35 years of age, fell in the street, and was brought up to the London Hospital, where a scalp wound was dressed. It was 10 a.m. when he entered the surgery. He was discharged after the

dressing of his wound, and at 5.30 p.m. he was again brought up to the hospital, this time by the police, who had found him insensible. On admission, he was unconscious and had stertorous breathing, the pulse was 72, laboured, the left pupil was larger than the right, but neither much dilated and neither reacting to light. The eyes diverged a little. There was a considerable bruise of the left eyelid, and on the occiput of the same side a considerable laceration. Mr. Hutchinson decided to trephine in an hour's time, but the man died before his return. At the autopsy the skull was found exceedingly thick and dense. Subdurally over the left hemisphere was a thick clot which weighed about five ounces, and extended to the base of the brain. A thin adherent film covered the whole of the opposite hemisphere, occupying the arachnoid cavity. There was no fracture of the skull, and no rupture of the middle meningeal artery. In the middle of the pons varolii were four or five well-marked circumscribed blood clots, from the size of a pea to a pin's head.

No. 60.—Mr. Jonathan Hutchinson, Senr. *Ibid.*, p. 49 (quoted in Mr. Jacobson's paper "On Middle Meningeal Hæmorrhage," under differential diagnosis).—A man fell from a hay cart in October, 1867. When admitted he had a cut on the occiput, with an irregularity in the bone near it. There was also a bruise on the right side of the head. He told his name and put out his tongue on admission, but soon afterwards became unconscious. He had left hemiplegia and left facial paralysis. The right pupil was widely dilated and not reacting, the left was normal. There was no stertor. He had been bled before Mr. Hutchinson saw him, probably causing the rapid and feeble condition of the pulse. There was a large extravasation of blood over the right temporal region. The diagnosis of ruptured middle meningeal artery having been made, the dura mater was exposed, but no extradural clot found. At the post-mortem examination, there was an extensive fracture of the occipital bone and posterior part of the parietal. A laceration of the outer surface of the right hemisphere was present near its middle, with much extravasation into the arachnoid. There was severe concussion of the whole brain.

No. 61.—Mr. George Cowell, Westminster Hospital Reports, vol. iv.—A man, about 45 years of age, was taken by the police to the surgery at 8.30 p.m. on October 1st in an unconscious condition. The police said that whilst intoxicated he had been knocked down by a cab. On admission, he was breathing slowly and heavily and could not be roused. When seen next morning by Mr. Cowell he was unconscious and breathing heavily. He could not be roused. There were slight abrasions on the head and face. No signs of fractured base and no vomiting. The jaws were somewhat rigidly fixed. The pupils were contracted, the left more than the right. Loss of power in the left arm with rigidity in right. There was very little difference between the legs, if anything the left was more rigid than the right. Both legs were slowly moved on tickling the soles. Complete retention of urine was present. Temperature 92°. Pulse 72. Respiration 24. The patient's symptoms gradually increased, the paralysis of the left arm became more marked on the morning of the 4th, and left facial paralysis showed itself. The unconsciousness deepened and the stertor became more marked. He died sixty-seven hours after admission. The temperature chart showed a gradual rise and subsequent fall. At the autopsy there was fracture of the base. On dividing

the dura mater a considerable layer of black clot was found covering the anterior part of the brain. The anterior part of the right frontal lobe was found lacerated and soft. The viscera were healthy.

No. 62.—St. George's Hospital Reports, vol. x., No. 1325.—D. M., a man of 48, was admitted to St. George's Hospital on August 9th. He had fallen downstairs whilst drunk and alighted on the back of his head. On admission, he was insensible and almost moribund from the loss of blood from two scalp wounds in the occipital region, which exposed bone freely. Stimulants being given, he revived. On August 10th to 12th he seems to have bordered on, if not actually suffered from, delirium tremens. On the 16th the scalp was incised for cellulitis, the temperature was 99°, the pulse 72. During the next three days there was some improvement. On August 21st there was rather less restlessness. Pulse 140. Temperature 103·4°. Complete right hemiplegia and coma. Death followed. At the post-mortem examination there was a fracture of the middle fossa on the right side. Over the left side of the brain there were 2½ of dark fluid and membranous coagulum. The under surface of the left frontal lobe was much bruised.

No. 63.—Drs. Homans and Walton. Boston Medical and Surgical Journal, vol. 124.—J. M., æt. 28, a waiter, was admitted into the Massachusetts Hospital after having been thrown from a horse and supposed to have struck his head against the cobble stones. On admission, temperature 98·2°. Pulse 60. There was a hæmatoma, with abrasion of the scalp, at a point corresponding to about the middle of the suture between the right parietal and occipital bones. The right pupil was dilated on admission. When seen by Dr. George Eliot, within two minutes of admission, consciousness was returning, the pupils were normal, the pulse slow and full. The hæmatoma was opened under cocaine but no fracture found. No paralysis. On the third day, May 15th, after admission he was delirious in the evening and trying to get out of bed. Temperature 103°. On the 16th he was almost comatose and aphasic. On the 21st the temperature had gradually come down to normal. The pulse was a little faster though still full and slow. Aphasic. Early in the morning of this day spasmodic twitchings of the right side of the face were noticed, and the right side of the mouth was drawn down. These recurred during the day. At 10 p.m. the same day convulsive movements appeared in the muscles of the left side of the face, and the right eyelid was kept closed whilst the left side was twitching. He seemed conscious though unable to speak. On the 10th day from admission, he had convulsive twitchings of the side of the face, increasing in frequency towards the evening; they commenced at the angle of the mouth. There was paresis of the right side of the face in between the attacks. The man was conscious, but aphasic. Operation was decided on. Before operation the convulsions had spread first to the right arm, then to the right side of the body, and finally to the whole body. Ether being given, trephining was performed just anterior to the lower part of the fissure of Rolando. The dura mater was bulging and tense, somewhat yellowish and opaque. Incision into it revealed a large clot, which was found to extend in every direction under the dura mater. The clot was removed. A small clot was also removed from underneath the pia mater by incision. The brain was lacerated to a considerable extent. No bleeding

point was found. The blood and clot were washed out with warm water. There were three slight convulsions affecting only the corner of the mouth the night after the operation, but no more occurred subsequently. The aphasia slowly but completely cleared up.

No. 64.—Dr. J. W. Elliott. *International Medical Magazine* (Philadelphia), 1893, vol. ii., p. 118.—A man, 30 years of age, was admitted to the Massachusetts General Hospital on November 25th, having been kicked by a horse three hours previously. He was unconscious for ten minutes after the injury. On admission, he was conscious, but could only speak with difficulty. The tongue was protruded to the left when put out. The pupils were equal and reacted. Pulse 96. No other paralyses. On the right side of the skull there was a compound depressed fracture. Ether was given and the depressed bone removed. The dura mater, which appeared white and uninjured, bulged into the wound and did not pulsate. Distinct fluctuations showed the presence of fluid under the dura mater. On incising the membrane a quantity of clear fluid escaped, but the pulsating brain was not visible. Another fluctuating membrane which had the colour of muscular tissue was pushed into the opening in the dura mater. On incising this membrane a little clear fluid and about an ounce of blood ran out. Pulsating brain convolutions then became visible for the first time. Blood continued to pour out of the subarachnoid space, and was only controlled by packing gauze deeply in between the convolutions. Recovery was complete.

No. 65.—Mr. Openshaw. *Abstracts of the Transactions of the Hunterian Society*, 1892-3, p. 36.—A man, æt. 60, fell from a cart whilst it was in motion. He walked into the hospital, where he exhibited considerable violence, and was with difficulty persuaded to remain in. There was a scalp wound two inches long on the left side of the occipital bone and profuse hæmorrhage from the left ear. After admission he became more and more unconscious and very restless. Two hours later there were no paralyses, consciousness was not wholly lost, the pupils were normal. Twenty-four hours after admission there was complete paralysis of left arm and leg, incessant movements of the right limbs, dilatation of the right pupil with failure to react to light, signs of subconjunctival hæmorrhage on the right side, paralysis of the right internal rectus. Two hours after this, subconjunctival hæmorrhage was appearing on the left side and stertorous breathing and coma were present. Trephining, without anæsthesia, was performed. On exposing the dura mater, the middle meningeal artery was intact, but the membrane bulged and was non-pulsating. A mass of blood-clot was found beneath, and partially removed. There was some improvement after the operation, but he sank and died twelve hours later. At the autopsy a large clot two and a half inches thick was present beneath the dura mater in the middle fossa on the right side. On the left side the petrous portion of the temporal bone was fractured, but no vessel of size seemed wounded.

No. 66.—Mr. H. W. Page. *Lancet*, 1898, vol. i., p. 1167.—The guard of a train was struck by an open door of a moving train. The blow caught him between the eyes, and threw him violently on to the back of his head. He was dazed but not unconscious, and in this condition was taken to St. Mary's

Hospital, at 1.15 p.m. on December 21st, 1897. On admission, he was extremely collapsed but perfectly conscious, answered questions, and gave his name and address. From the left ear there flowed blood and cerebro-spinal fluid. Respirations 20, pulse 90. No fracture could be discovered, but there was some slight swelling in the left temporal region. The pupils were equal and reacted sluggishly. There were no unilateral symptoms save the aural hæmorrhage. Soon after admission he passed into coma. Between the time of admission and the time at which he was seen by Mr. Page (2 p.m.) he had some clonic movements of the left arm, but these had quickly given way to spastic rigidity, which quickly involved all the limbs equally. The coma deepened, the pulse and respiration began to fail, the pupils became unequal, the left being much larger than the right, and it was decided to operate. He was trephined over the left middle meningeal artery; the dura mater bulged, was bluish in colour and non-pulsating. On incision, a considerable amount of blood flowed away. As the blood was thought to be chiefly at the base of the brain, an irrigator was passed down to this part. There was marked improvement in the pulse and respiration, and the left pupil contracted down to the same size as the right, but the man never regained consciousness, and died six hours later, the temperature having steadily risen to 107° C. before death. At the autopsy the subarachnoid space over both hemispheres was practically full of blood, both fluid and clotted, but the greatest quantity of blood was at the base. There was extensive laceration of the left orbital lobe, and anterior extremities of both temporo-sphenoidal lobes. There was a most extensive basal fracture.

No. 67.—Dr. J. Ramsay. *Inter-Colonial Medical Journal of Australasia* (abstract in the *Journal of the American Medical Association*, January 26th, 1901, p. 261).—A man of 54, addicted to alcoholic excess, was found unconscious in the road, having probably fallen from his horse. In the course of an hour or two later the man partially recovered consciousness and became greatly excited. Forty hours later he had irregular movements and twitchings in the right wrist and hand. Later these affected the left wrist and hand, then the lower extremities, and finally the left side of the face. General convulsions followed, starting on the left side of the face and extending to left arm, left leg, and then the right side. The right side of the face was not involved. In the intervals the right arm was rigid and the left flaccid. The urine was albuminous. The breathing became Cheyne-Stokes in character. He was trephined over the right motor area, and a large flat blood clot found on the surface of the brain beneath the dura mater. This was removed and the cavity left drained with gauze. Two convulsions occurred subsequently, but no more. Some mental and motor impairment remained though lessening in degree.

No. 68.—Sir Benjamin Brodie. *Medico-Chirurgical Transactions*, vol. xiv. p. 327.—A man, 35 years of age, fell out of a cart and struck his head on the pavement on November 8th. He was bled by a practitioner in the neighbourhood and afterwards taken to St. George's Hospital, where he talked and reeled like a drunken man. He was again bled. On November 9th he had headache but was otherwise well. He continued so until November 12th at 5 a.m., when some of the patients in the same ward heard him talking

incoherently. The house-surgeon was called and found the man insensible, motionless, with stertorous breathing and dilated pupils. He was bled from the arm without effect, and died half an hour after the commencement of the symptoms. At the post-mortem examination a thin layer of blood was found extravasated between the arachnoid and pia mater over the posterior part of the two hemispheres. In the lower part of the anterior lobe of the cerebrum the substance of the brain had been ruptured, and between the dura mater and the arachnoid was about two and a half ounces of blood.

No. 69.—Dr. Lucius W. Hotchkiss. *Annals of Surgery*, 36, p. 82.—D. McG., æt. 39, fell backwards down some stairs, a distance of about nine feet, striking the back of his head. He was picked up unconscious and remained so for eight hours. When he recovered consciousness he appeared dazed and had difficulty in speaking. Also said to have bled from the right ear. He was able to walk up to hospital and was admitted. There were no paralyses, but motor aphasia was present. Temperature 101°; respiration 20; pulse 90. The urine contained a trace of albumen. There was a small abrasion over the right side of the occiput. On the 9th facial paralysis was noted. Temperature 102° at noon. On the 10th some bleeding from the right ear set in in the early morning. At 8 a.m. the temperature was 101·6°, the pulse 68, the motor aphasia was practically complete, the grip of the right hand was feeble, and there was right facial paralysis. Ether was given and trephining over the left middle meningeal artery performed. There was no evidence of fracture. Some dark fluid blood was removed from beneath the dura mater with an aspirator after which the dura mater was opened and the clot exposed. This was firm and moderately adherent to the cortex of the brain. It was washed away with a stream of salt solution. Slight cortical laceration present. Temporary drainage employed. Two slight convulsions occurred after the operation, but his recovery was rapid and complete.

No. 70.—Dr. Graeme M. Hammond. *Journal of Nervous and Mental Diseases*, December, 1899 (taken from the *Lancet*, February 24th, 1900, where the case is abstracted).—A clerk, 25 years of age, of temperate habits, who had suffered for some weeks from albuminuria and granular casts, was struck on the left temple with a loaded whip handle in a street fight. He was knocked down, but did not lose consciousness, and was able to get up and walk home, though a few days later he was seized with an attack of general convulsions of a severe and prolonged character. On recovery of consciousness it was found that he had completely lost the power to name objects and persons. Ophthalmoscopic examination showed slight choking of the right disc. A diagnosis of subdural hæmorrhage over the posterior part of the left superior temporal convolution was made. At the operation (by Dr. Seneca Powell) two weeks after the injury, a linear fracture was found in the left temporal bone, and a subdural blood clot covered the entire superior temporal gyrus. In the posterior part there was a hole into which a probe could be passed one and a half inches. The cerebral tissues were here lacerated, probably by indirect violence. Patient made a good, but not complete recovery.

No. 71.—Dr. George Tully Vaughan. *American Journal of Medical Sciences*, vol. 122.—K. M., æt. 18, a white man, was admitted to the Emergency Hospital, on August 10th, 1899. He had been struck on the head with a club and knocked down. On being assisted to his feet he walked a short distance, fell again, and lapsed into coma. Examined an hour after his injury, he was found profoundly unconscious, breathing deep, sometimes sighing, both pupils dilated and immobile. Pulse 60, full. Clonic contractions at short intervals of the right arm and leg and sometimes the left arm, with persistent tendency of the face to turn to the left side, were found. There was a small contused wound of the scalp not extending to the bone just above the left ear. Trephining, without an anæsthetic, was performed over the left fissure of Rolando. The dura mater was found bulging and tense. A large black clot was found beneath it about the size of an orange. A freely bleeding artery on the surface of the brain was ligatured. No pulsation in the part of the brain exposed was observed. The only change after the operation was contraction of the pupils and diminution in the frequency of the convulsions, the patient dying two hours later without return of consciousness. The necropsy revealed numerous small hæmorrhages throughout both hemispheres.

No. 72.—Mr. Golding-Bird. *Guy's Hospital Reports*, vol. xxx.—Thos. D., æt. 31, admitted into Cornelius ward May 22nd, 1888, suffering from concussion. He had fallen from a cart one hour before. He vomited on the way to the hospital. At 1 p.m., when he was admitted, he could be made to answer questions after a fashion, but relapsed at once into irritable stupor. Later he became very violent, and passed into coma soon after 8 p.m. Mr. Golding-Bird was called to him at 10 p.m. and found him comatose, with stertorous breathing, limbs and face paralysed, pupils inactive and dilated, the left more so than the right. The left side of the face appeared slightly more paralysed than the right. Pulse 116; Temperature 102·8°. No wound and no signs of fractured base. Over the right parietal bone the scalp was bruised and puffy, with extravasated blood. Pressure over the bruised area caused extension movements of the left forearm and wrist, and greater pressure led to convulsive movements of the left arm and leg. No anæsthetic was given, but the skull was trephined on the right side just anterior to the right parietal eminence. A short fissure of the skull was found. An extradural clot was present. As the dura mater was bulging it was tentatively incised. A stream of dark blood immediately rose to a height of half an inch. A slight improvement in the breathing followed, but he died two to three hours later. At the autopsy the only fracture found was the one found at the operation. In the arachnoid cavity, chiefly on the left side, were three and a half ounces of blood. The left temporo-sphenoidal lobe was much bruised on its anterior and under surface.

Since writing this paper, my attention has been called by Mr. Mills, Librarian to Guy's Hospital, to a paper in the *American Journal of Medical Sciences* for April, 1895, on *Meningeal Hæmorrhage*, by Drs. Scudder and Lund. In this

paper twenty-one cases of subdural hæmorrhage were reported. Seven of these twenty-one cases are included in the seventy-two cases upon which this paper is based. References to the remaining fourteen I give below. An examination of these cases in no way affects the conclusions arrived at in the above paper.

References to cases published in Drs. Scudder and Lund's paper and not reported above :—

1. Case 1.—Schneider. *Archiv. für Klin. Chir.*, 1887.
2. Case 2.—Ceci. *Deutsch. Med. Zeitung*, 1887.
3. Case 5.—Ball. *B.M.J.*, 1888.
4. Case 6.—Walker. *Medical Age*, 1888.
5. Case 8.—Matas. *New Orleans Med. and Surg. Journ.*, 1889.
6. Case 10.—Mills. *Journ. Nerv. and Mental Disease*, 1890.
7. Case 11.—Chiéne. *Transactions American Surg. Assoc.*, 1891.
8. Case 12.—Duret. *Cong. Trans. de Chir.*, Paris, 1891.
9. Case 14.—Elliot. *Massachusetts General Hospital Records*, Vol 281, p. 41.
10. Case 15.—Beach. *Ibid.*, Vol. 274, p. 43.
11. Case 16.—A. T. Cabot. *Ibid.*, Vol. 257, p. 214.
12. Case 17.—C. B. Porter. *Ibid.*, Vol. 259, p. 136.
13. Case 20.—Mynter. *Annals of Surgery*, 1894, p. 19, 539.
14. Case 21.—Walker. *Cincinnati Lancet-Clinic*, June 17, 1893.

CASES OF SUBDURAL HÆMORRHAGE

Thought to be due to injury, but in which the lesion was found at a post-mortem examination long after :—

1. *Transactions of the Pathological Society* (vol. ii., pt. 2, p. 172).
“Fracture of the base of the skull, after which the patient lived two months.”—Mr. Gray.
2. *Ibid.* (vol. vi., p. 5) :—“A false membrane lining the smooth surface of the dura mater, covering the right cerebral hemisphere, probably the result of extravasation of blood from an injury.”—Dr. Ogle.
3. *Ibid.* (vol. vi., p. 8) :—“Large cyst from the cavity of the arachnoid.”—Dr. Quain.
4. *Ibid.* (vol. xxxvi., p. 16) :—“Hæmatoma of the dura mater.”—Dr. F. Charlwood Turner.
5. *Ibid.* (vol. xlviii., p. 10) :—“Hæmatoma of the dura mater with development of membrane.”—Cyril Ogle, M.B.
6. *Transactions of the Medico-Chirurgical Society* (vol. xxviii.) Prescott Hewitt's paper on “Extravasations of blood into the cavity of the arachnoid.”
7. Case mentioned on p. 598 of Bowlby's *Pathology*.

REFERENCES TO PATHOLOGICAL SPECIMENS OF
SUBDURAL HÆMORRHAGE.

Through the courtesy of the Curators of the Museums of St. Bartholomew's, London, and St. Thomas's Hospitals, I was enabled to examine the museums of these hospitals with a view to finding specimens of subdural hæmorrhage the result of injury. My search was not very successful, for in five of the largest museums, including the three above, our own, and that of the Royal College of Surgeons of England, I only found one good example of this condition. This was specimen No. 2448 in the museum of St. Bartholomew's Hospital, which is described as "A large clot of blood adhering to the internal surface of the dura mater which covered the upper part of one of the hemispheres of the cerebrum. The effusion was in consequence of injury." The only other specimen of the condition which I found is No. 2446a in the same museum, which is described as "Portions of left parietal and temporal bones showing a fracture running across the line of the middle meningeal artery. The artery is torn across and some blood-clot lies in the groove in the bone. From a man, æt. 64, who fell off his cab and was admitted into the hospital drowsy and helpless. From this condition he rallied and after ten days was able to sit up in a chair; on the thirteenth day from the injury he suddenly became comatose and died. A post-mortem examination showed a hæmorrhage into the arachnoid cavity, some of the blood being recent whilst the smaller portion evidently dated back to the time of the accident. The vessels were atheromatous."

In the same museum there is a specimen of subdural hæmorrhage in a case of cirrhosis of the liver (No. 2447 b) and one of subarachnoid hæmorrhage in a case of malignant endocarditis (No. 2470 a).

Specimen No. 1978 in the museum of St. Thomas's Hospital is a "large subdural clot due to rupture of a small aneurysm of the middle cerebral artery," from a patient æt. 51, who lived for a week with hemiplegia, hemianæsthesia, and aphasia, after being taken suddenly ill with sickness (Reported by Dr. Sharkey, Pathological Society's Transactions, vol. 27, p. 3).

In the Guy's museum specimens, Nos. 1591⁷⁰, 1591⁸⁰, 1592¹⁰, 1605⁸² and 1605⁸⁸, are cases of old hæmorrhagic membrane or cyst in most cases of unknown origin.

THE BEHAVIOUR OF LEUCOCYTES UNDER THE INFLUENCE OF CERTAIN BACTERIAL AND OTHER SUBSTANCES.

BY THOMAS EDWARD HOLMES, M.D.

(Thesis for the M.D. Cambridge.)

THE following experiments were undertaken at the instigation of Dr. Ainley Walker, with the object of determining, if possible, the reaction of the leucocytes of rabbits' blood to various bacterial (dead and living cultures) and other toxic products, and also to ascertain whether the different forms of leucocyte reacted constantly with the same kind of injection.

It is only within recent years that the study of leucocytosis, both from a clinical and experimental aspect, has attracted much attention.

As early as 1864, it was shown by Traube and Geschleiden that the animal body quickly disposes of bacteria introduced into it by artificial means, but it was not until Ehrlich's valuable discovery of the differentiation of leucocytes by the aniline dyes, that the histology of the blood was placed on a firm basis. Numerous theories have been advanced to explain the germicidal power of blood, but, in the light of modern work, no explanation which is not open to serious objection was suggested until Metchnikoff brought forward his cellular or phagocytic theory.

This eminent observer was, however, compelled to admit in 1889 that at least part of the bactericidal action *in vitro* was attributable to substances present in the serum, which he claimed were yielded to it by the leucocytes in the process of coagulation. On this question, a large amount of work has been done, commencing with the observations of Hankin and Kanthack in 1892, and subsequently carried on by Loewy and Richter, von Fodor, Denys, and Lecelf, Havet, Buchner, Bordet, and many others. These observers have shown by their results that:—

1. The fresh blood of an immune animal possesses a bacteriolytic action which is greatest when the animal concerned presents a condition of marked leucocytosis, and is diminished when the leucocyte count falls below the normal.

2. Sterile pleural exudates rich in white corpuscles possess high bacteriolytic power, but if these corpuscles be rapidly separated from the fluid by filtration, the bacteriolytic action disappears, and is at once restored on their replacement. If, on the other hand, the leucocytes are merely broken up and disintegrated by alternate freezing and thawing, the germicidal power remains.

To explain these phenomena, Bouchard advanced the cellulo-humoral theory, which involves the simultaneous action of two forces—the bactericidal power of the serum, and the phagocytic activity of the corpuscular elements. This cellulo-humoral theory in its modern form, forms to-day the only really tenable position on the general question. I have somewhat digressed from the main subject of experimental leucocytosis, but the above brief account of the latest opinions on immunity does not seem out of place in a paper on at least one important factor, the rôle of the leucocytes, in the destruction of bacteria and other toxic substances. All observers are agreed that bacterial and toxic inoculation in animals produces a marked effect on the circulating leucocytes. Immediately following such inoculation, we have the stage of diminution of total leucocytes to a greater or lesser degree, the stage of leucopenia, or hypoleucocytosis; this is only temporary, and is followed by a great increase in the total number of circulating leucocytes, the stage of hyperleucocytosis, or simply leucocytosis. Among the various

substances that have produced these phenomena on injection may be mentioned:—

Hæmoglobin.	Septic fluid.
Pus.	Lymph cells.
Hemialbumose.	Peptone.
Pepsin.	Nucleic acid.
Nuclein.	Leech extract.
Tuberculin.	Pyocyanin.
Curare.	Uric acid.
Urates.	Carmine in suspension.
Dead bacterial products.	Living bacterial products.
Dead bacterial proteins.	Filtered yeast cultures.
Tincture of Myrrh.	Extract of Gentian.
Sodium cinnamate.	Animal cells, etc., etc.

The stage of leucopenia has been practically neglected in the present paper, as all blood-counts have been made at least fifteen to twenty hours after inoculations; but it will be seen from the charts that even as late as this, there was frequently a smaller number of circulating leucocytes present than in the blood-count previous to injection.

According to most authorities leucopenia is well established within a very short time of inoculation. In some of Lowit's experiments, the number of leucocytes fell in five seconds from the time of injection to 1–20 of the number circulating before inoculation. In some instances, on the other hand, no diminution of leucocytes has been observed, the total number gradually increasing from the time of injection.

Schlesinger, in a lengthy monograph on the subject, observed a leucopenia usually two hours, but never later than four or five hours after inoculation. He, however, admits that great variations occur, and that no definite rule can be made. Most observers agree that the polynuclear leucocytes disappear in the stage of leucopenia, while the mononuclear forms are scarcely affected, but Schlesinger says the lymphocytes are preponderately involved, and Muir maintains that all varieties are affected. Sherrington, in some experiments on inflammatory leucocytosis, noted that the coarsely granular cells disappeared almost entirely in

hypoleucocytosis, and that in the subsequent hyperleucocytosis the finely granular leucocytes were strongly in evidence. Milroy and Malcolm observed the same phenomena both in the blood and marrow after injections of nucleic acid. Durham concludes that the coarsely granular eosinophil cell takes a minimal share in the process of bacterial destruction. Lowit thought the polynuclear leucocytes were destroyed (dissolved up) during hypoleucocytosis. Rieder, on the contrary, maintained that they were temporarily collected in some region of the circulation, which they left after a time to become again distributed through the circulation, when a substance inducing positive chemiotaxis reappeared in the blood. Werigo suggested that the leucocytes crowded into the lungs, liver, and spleen after the intravenous inoculation of particulate material. Goldscheider and Jacob have proved this for the lungs, and Everard, Demoor, and Massart pointed out that the medulla of bone must also be included. Werigo says the injected material is enveloped by the white corpuscles, the latter being arrested in the liver, where they transfer the particles to the endothelial cells of that organ; the phagocytic cells of the splenic pulp have similar functions. Roger and Josué have noted a proliferation of "celles medullaires" after injections of phosphorus, etc. Haushalter and Spillman have also observed important changes in the marrow after toxins of bacillus coli communis and staphylococcus. Muir, in a recent article, has shown that in rabbits, the bone-marrow is the site of multiplication of both the finely and coarsely granular polynuclear forms.

According to Schlesinger, hyperleucocytosis is often the only symptom of the infection. In exceptional cases the polynuclear leucocytes are increased. In Bullock's experiments on rabbits with sodium cinnamate, an almost pure polynuclear leucocytosis was obtained, and Batty Shaw observed the same phenomena with similar injections in cats. The increase takes place usually on the second or third day after injection (6,000 to 15,000 above the normal) and reaches its maximum on the third or fourth day. The subsequent fall to normal is gradual; in one of Schlesinger's experiments it was not reached until the fourteenth day.

Hyperleucocytosis is not influenced by fever or changes in the local infection. This was also pointed out by Sherrington and Nicolas and Courmont.

Besredka, from a large number of experiments with diphtheria toxin, concludes that:—

1. In intoxication by large doses, the polynuclear leucocytes describe a curve having a parabolic form, and rise to a maximum ten to sixteen hours after inoculation. They decrease regularly and rapidly until death.

2. In intoxications which slowly kill in several days, the course of the leucocytes is represented by a curve with oscillations "*assez étendues*," having for essential characters—

- a.* De se maintenir toujours au dessus du taux normal.

- b.* De ne s'interrompre à aucun moment de l'intoxication.

3. The animal intoxicated with large doses of toxin, and saved by serum, remains ill "polynuclearly" during twelve to fifteen days, during which the same oscillations as in the slow infection occur, but with this difference, that this time, the leucocytes end "*par prendre le dessus ce qui se traduit par un retablisement progressif et graduel du chiffre normal.*"

I have given these conclusions in detail, as they bear very closely on a number of similar experiments in this paper. The polynuclear hyperleucocytosis has been noted by numerous observers in a variety of experimental inoculations, but I have been unable to find any precise numerical observations on the mononuclear leucocytes, with the exception of those of Bullock and Muir.

Achard and Loeper observed a constant polynuclear leucocytosis after bacterial injections until the third or fourth day, when the polynuclear leucocytes diminished, and "the mononuclear leucocytes increased in number." Muir often noticed an increase of hyaline leucocytes at a later stage, and suggests that the late mononuclear reaction may be due in part to chemiotactic substances derived from broken down finely granular leucocytes. Besredka says that in the course of immunization against diphtheria toxin, each injection of the latter is followed by a hyperleucocytosis entirely dependent on polynuclear leucocytes,

but as immunization proceeds the polynuclear forms are less prompt to act and are replaced by mononuclear leucocytes. Schlesinger states that the lymphocytes may sometimes increase very considerably in the later stages of hyperleucocytosis.

When a fatal issue ensues after inoculation, the leucocytes behave somewhat differently. If death occurs after several days the hyperleucocytosis is not appreciably different from the condition previously sketched, and which occurs in cases ending in recovery (Schlesinger). In this observer's experiments, at the time of death, the total number of leucocytes had again fallen to normal for one or two days, but on a few occasions the hyperleucocytosis was still at its maximum. I shall refer to this again at a later stage. Schlesinger suggests that the increase of leucocytes at death may be either active and chemiotactic, in which case the polynuclear leucocytes are increased, or passive and purely physical, with an increase of mononuclear leucocytes. Gabritchewsky concludes that inoculations of entire cultures of *bacillus diphtheriæ* producing death, are followed by a hyperleucocytosis, which increases progressively till death; on the contrary, with an immunized animal, the hyperleucocytosis reaches its maximum eight hours after injection and disappears completely at the end of twenty-four hours. Sherrington, experimenting with cats, noticed that death was preceded by a stage of leucopenia. In some of Schlesinger's fatal cases, there was a gradual diminution of total numbers from the time of inoculation until only 2,000 per c.cm. or even fewer leucocytes were present just before death. In these instances, the same observer points out that the polynuclear reaction is small and transient.

Everard, Demoor, and Massart also observed the same phenomena in subcutaneous injections of hog cholera, producing death in twenty-two and a half hours, and in intravenous injections of *bacillus anthracis* causing death in twenty hours.

Various theories have been adduced to explain these phenomena. Romer suggested that the increase of leucocytes was due to a rapid multiplication in the veins, as well as in the blood-forming organs, but Kanthack showed that venous blood did not contain

any more leucocytes than arterial blood. Lowit also pointed out well-founded objections to Romer's views, and thought that the leucocytosis was due to an increased supply of young leucocytes to the blood, which at a later stage developed into polynuclear leucocytes. He supposed that this excessive production was due to chemical stimulation of leucocyte-forming organs by substances shed into the blood-plasma at the time of disintegration of the hæmic leucocytes.

Bruce, Goldscheider and Jacob, Muir, and others, however, have clearly shown that the leucopenia occurring after injections of such substances as nucleic acid, peptone, bacterial products, extracts of various organs, etc., is due to the accumulation of leucocytes in the capillaries of certain organs, especially the lungs, liver and spleen. The leucopenia is also due, to some extent, to emigration of leucocytes to the site of inoculation, as pointed out by Muir. The latter observer, with Ehrlich, Goldscheider and Jacob, considers that chemiotaxis is the all-important agent in the production of leucocytosis. The causation of the latter within so short a time of inoculation, has been much debated in recent years.

Ehrlich, working with Lazarus, states definitely that leucocytosis is a function of the bone marrow, and Goldscheider and Jacob assert that the rapid occurrence of leucocytosis is due to the passage of leucocytes from the bone-marrow to the circulating blood.

Muir is unable to speak definitely as to the mechanism by which the increased proliferation is brought about. He says, "The passage of leucocytes from the bone-marrow to the blood may in itself be sufficient to produce increased multiplication of the mother cells. The newly-formed cells will be constantly passing into the blood, and on the principle of over-regeneration after repeated loss, as expounded by Weigert, a hyperplasia of myelocytes may result; on the other hand, it is possible that the substances which act chemiotactically may also directly stimulate cellular proliferation."

Ehrlich, Engel and Ribbert assert that the granular and large mononuclear leucocytes are formed in the bone-marrow, while

the lymphocytes have their origin in the lymphatic glands; these observers also say that the large mononuclear forms may be formed in the coelomic cavities. Myers says that the large lymphocytes are capable of being transformed into transitional cells, and admits that some polynuclear leucocytes are manufactured in the blood-stream from large mononuclear forms. Muir, on the contrary, contends that there is no evidence of the transformation from the non-granular to the granular type in the circulating blood, and has clearly shown that the bone-marrow is the site of multiplication of the granular leucocyte. Gulland, Arnold, Uskoff and Frankel maintain that all leucocytes are derived from small lymphocytes, and Everard, Demoor, and Massart conclude that the different forms of leucocytes are evolutionary stages of the same cell:—

1. Single nucleated cell with little protoplasm.
2. Vesicular nucleus with more protoplasm.
3. Adult polynuclear leucocyte with phagocytic functions and protoplasm loaded with granules.

According to Councilman, Mallory and Pearce, the large mononuclear leucocytes are derived from the endothelium of lymph and blood vessels and coelomic cavities, and this conclusion is the one most generally accepted at the present time.

METHODS.

For the purposes of these experiments the diet of the animals concerned was very carefully regulated. All edible material was removed from the cages at 6 p.m. every evening and the blood taken from the ear early each morning before the first meal of the day was given. The diet consisted entirely of green food and dry biscuits, to which the animals had become accustomed for some time before inoculation. Under this treatment they remained in good condition and increased in weight when not the subjects of bacterial infection. Three months were spent in acquiring familiarity with the leucocytes of rabbits' blood under both normal and experimental conditions. During this period,

my results were daily checked by Dr. Ainley Walker until sufficient accuracy had been gained. These observations, which were regarded as preliminary, have only been recorded in relation to the total number of leucocytes. The subsequent observations were also checked from time to time by Dr. Ainley Walker, and in all cases the two results were found to agree very closely. The rabbits selected for inoculation were in every instance well grown and healthy, and special precautions were taken to ensure a normal condition by counting the blood for several days (in some cases a week) before inoculation. If a leucocytosis was found to be present the animal was discarded.

The Thoma-Zeiss apparatus was used for the estimation of the leucocytes, and the leucocytes in the whole number of squares on the counting chamber were counted. Never less than four such counts were made of each diluted drop of blood, and frequently where small variations were found to exist, as many as eight or ten separate drops of the diluted blood were counted. The blood was obtained by pricking the lateral vein of the rabbit's ear with a flat V-shaped needle with both edges sharpened. This form of needle makes a very small wound, and one which rapidly heals. The larger sized pipette was used for diluting the blood, and in the earlier experiments, Toisson's fluid, prepared according to the following formula, was the diluent:—

Distilled water	160 c.c.
Neutral glycerine (30° Baumé)	30 c.c.
Pure Sodium Sulphate	gm. 8.
" " Chloride	gm. 1.
Methyl Violet 6 B	gm. 0.25.

Dissolve the methyl violet in the glycerine with half the water added. Dissolve the salts in the other half of the water. Mix and filter. Filter before use.

This stain was employed for estimating the number of the red and white corpuscles, but subsequently for the enumeration of the leucocytes, a two per cent. solution of acetic acid, with just sufficient gentian violet to stain the nuclei, was found to give better results. In the case of the red blood corpuscles, the number in sixteen squares was counted, and an average taken

per square. The blood was always counted by the wet method as soon as possible after it had been obtained. Throughout the experiments, blood-counts were made daily, an interval of twenty-four hours separating each count. A dilution of 1 in 100 was used for all enumerations of the leucocytes.

The film preparations were stained with Jenner's fluid, but I found that three to five minutes was not long enough to stain the majority of the films; frequently the latter were left in the stain for half an hour or even longer. I tried several brands of Ehrlich's triacid stain, but was unable to obtain sufficiently good results.

CLASSIFICATION OF LEUCOCYTES.

I have made no attempt in these experiments to give a detailed account of every form of leucocyte met with, since the wet method of enumeration has been used almost exclusively.

Two main varieties have been distinguished:—

1. *Polynuclear leucocyte* with the nucleus in several masses united by relatively slender bridges of nuclear material, and a granular protoplasm.
2. *Mononuclear leucocyte* with a single symmetrical nucleus and non-granular protoplasm.

This variety includes large and small lymphocytes with a relatively large and rounded nucleus, and very little protoplasm, and large mononuclear leucocytes with a rounded or horse-shoe shaped nucleus, and more protoplasm than the former.

About one hundred films were counted differentially with 1-12 oil immersion Leitz objective, with the object of determining, if possible, some indications of the function of the different varieties of the mononuclear leucocyte. The results obtained by counting one hundred mononuclear forms in each film are given in the tables later, but from the few observations I have made, it does not seem advisable to draw any definite conclusions.

In this differentiation by the film method, I have distinguished three forms:—

1. *Small lymphocyte*.—This is generally about the size of a red corpuscle, with a large rounded nucleus occupying the greater part of the cell. In some of the experiments where a mononuclear leucocytosis was present a great number of very small lymphocytes were seen. The nucleus stains somewhat indistinctly with Jenner's fluid, while the protoplasm takes the stain deeply.
2. *Large lymphocyte*.—This cell has the same essential characteristics as 1, with the exception that it is larger, always having a bigger diameter than the red corpuscle.
3. *Large mononuclear leucocyte*.—This is the mononuclear phagocyte of Metchnikoff. It has a fairly abundant non-granular protoplasm, and a nucleus which may be rounded, horse-shoe shaped, oval, etc.

DESCRIPTION OF NORMAL LEUCOCYTES OF RABBITS' BLOOD.

Brinkerhoff and Tyzzer, in a recent paper on the leucocytes of normal rabbits' blood, give the following classification :—

Polynuclear leucocytes :—

1. With affinity for basic stain—mast cells.
2. " " acid "—amphophiles.
3. " " eosin "—eosinophiles.

Mononuclear leucocytes :—

1. With strong affinity for basic stain—lymphocytes.
2. " weak " "—large mononuclear leucocytes.

These varieties occur in the following percentages :—

Amphophiles	40 to 50 per cent.
Eosinophiles	5 " 1 "
Mast cells	4 " 8 "
Lymphocytes	45 " 55 "
Large mononuclears	2 " 8 "

Muir says the lymphocytes number 30 to 40 per cent., but may be as numerous as the amphophiles.

Brinkerhoff and Tyzzer maintain that the following factors must be considered in drawing inferences from variations in the leucocyte count :—

1. Loss of body heat.
2. Shock.
3. Fasting and feeding.
4. Pregnancy.

All these factors have been duly considered in the present paper. In a large number of normal rabbits in Brinkerhoff's and Tyzzer's paper, the leucocyte counts varied between 4,600 and 13,400.

Bullock says the normal number varies between 6,000 and 9,000, but I agree with the former observers that a wider limit than this exists in the healthy rabbit.

INJECTIONS.

The following is a list of the various injections used in my experiments :—

1. *Diphtheritic toxin*.—This was a specimen of diphtheritic toxin which was kindly supplied to Dr. Walker by Dr. Vernon Shaw, of the Brockwell Park Laboratories. It was derived from cultures of the bacillus diphtheriæ Americanæ of Parkes.
2. *Vibrio Metchnikovii*.—This was an old laboratory culture of this micro-organism.
3. *Bacillus typhoidæ abdominalæ*.—A stock laboratory culture of low virulence, and in constant use in the bacteriological department for carrying out the Gruber-Durham reaction. In the earlier injections of 2 and 3 of agar cultures, the growth was washed off the surface of the agar with ordinary culture bouillon; in the later injections normal salt solution was employed for the same purpose.

4. *Extract of tubercle bacilli*.—This was an ethereal extract of dried tubercle bacilli which was brought into suspension, after the ether had been driven off, in a solution of sodium hydrate having an alkalinity of .2 per cent., i.e., the alkalinity of normal blood. The extract was further sterilized by the discontinuous method on five successive days before inoculation.
The tubercle bacilli, which were kindly supplied by Professor McFadyean, of the Royal Veterinary College, were treated according to the method described by Aronson, who extracted a fatty acid and wax from large quantities of bacilli by treatment with alcohol and ether.
5. *Extracted tubercle bacilli*.—These had been twice extracted with boiling ether, and subsequently with a mixture of ether and absolute alcohol. They were obtained from the same source as 4.
6. *Animal cells*.—Red corpuscles were prepared for inoculation from defibrinated sheep's blood, which had been collected with aseptic precautions, and subsequently tested for sterility by centrifugalization and repeated washing of the corpuscles with normal (0.85 per cent.) salt solution. Renal, ovarian and testicular cells were prepared by macerating the organs, which had been previously cut into small pieces, in a saturated solution of urea according to the method described by Ramsden. The material was then rubbed up in a mortar and freed as far as possible from urea by repeated washing and centrifugalization. It was then suspended in a few c.c. of normal salt solution, strained through a piece of sterile muslin to remove connective tissue, etc., and injected into the peritoneal cavity of an animal.
7. *Broth*.—Ordinary sterile culture bouillon.
8. *Bacillus diphtheriæ Americanae*.—These were agar cultures suspended in normal salt solution.
9. *Staphylococcus pyogenes aureus*.—An old laboratory culture of staphylococcus aureus. Agar cultures were washed off the surface of the agar with normal salt solution and so injected.

10. *Dead tubercle bacilli* from the same cultures as those previously mentioned.
11. *Micrococcus rheumaticus*.—This was an organism which was isolated by Dr. Ainley Walker from a case of rheumatic fever. It had given rise to acute rheumatism in several rabbits. The injections used were from agar or blood-agar cultures.
12. *Pneumococcus*.—This was a highly virulent organism kindly supplied by Dr. Eyre. For the purpose of the present investigation it was attenuated by growth in ordinary bouillon and frequent re-inoculation in sub-culture during several weeks.
13. *Sheep's serum*.—Sterile serum obtained from sheep's blood which had been collected with as strict aseptic precautions as possible, from the slaughterhouse, in sterilized flasks, immediately whipped and conveyed to the laboratory.
14. "*Peptone*." — Commercial peptone containing approximately 95 per cent. of albumoses.

ABSTRACT OF EXPERIMENTS.

REACTIONS TO VIBRIO METCHNIKOVII.

Rabbit A. Chart 1.—Two agar cultures seventy-two hours old of living vibrio Metchnikovii injected subcutaneously affected both varieties of leucocyte, but more especially the polynuclear forms. Both kinds reached a maximum (polynuclears, 11,000; mononuclears, 7,000) on the third day, and then gradually decreased in numbers. There was a slight diminution of both varieties in the early stages.

Three agar cultures twenty-four hours old of living vibrio Metchnikovii were again injected fifteen days after the first inoculation. There was a marked fall in both forms for two days, when they began to increase in number. The number of polynuclear leucocytes was highest on the seventh day (10,000 per c.mm.) and that of the mononuclear leucocytes on the eleventh day (10,000 per c.mm.).

Rabbit B. Chart 3.—The injection of one agar culture twenty-four hours old of living vibrio Metchnikovii subcutaneously, gave a hyperleucocytosis entirely confined to the polynuclear leucocytes. An increase of these was noted on the third day of 7,000 per c.mm. The mononuclear leucocytes were diminished throughout the reaction.

Rabbit H. Chart 11.—One agar culture twenty-four hours old of living vibrio Metchnikovii injected subcutaneously, produced a marked increase in the polynuclear leucocytes, the latter reaching a maximum of 11,250 per c.mm. on the third day after inoculation, and quickly falling again to normal. The mononuclear forms did not increase until the fourth day, when they were as high as the polynuclear leucocytes. Their fall to the normal was more gradual. *Cp.* Everard, Demoor and Massart.

A guinea-pig of 850 grammes was injected subcutaneously with 3.5 c.c. of four days old vibrio Metchnikovii which had been heated to 100°. There was a polynuclear leucopenia within an hour, and a hyperleucocytosis two hours after inoculation. Nineteen hours afterwards the polynuclear leucocytes were predominant, and a very large increase of the same kind was noted at the twenty-fifth hour, the mononuclear leucocytes being at this time very rare. Forty-nine hours after inoculation, the polynuclear leucocytes were largely predominant, but the mononuclear forms had increased. 5 c.c. of the same culture were also injected subcutaneously into a guinea-pig of 1,020 grammes. No diminution of leucocytes was observed, and the hyperleucocytosis was almost entirely polynuclear.

It will thus be seen that these injections of living cultures of vibrio Metchnikovii produce an increase in both forms of leucocyte, but more especially the polynuclear forms.

REACTIONS TO ANIMAL CELLS.

Sheep's red corpuscles.—Rabbit D. Chart 5.—10 c.c. injected intraperitoneally produced a diminution of both varieties for the first few days. The mononuclear leucocytes rose again on the fifth day after inoculation to 7,000 per c.mm., having been about

4,000 for the four previous days. The polynuclear leucocytes were only slightly increased; the greatest number recorded during the experiment was 4,500 per c.mm.

A similar but larger injection (25 c.c.) was given subcutaneously in rabbit D, Chart 6, after an interval of nineteen days. In this experiment there was an almost pure mononuclear leucocytosis, as the polynuclear leucocytes were only increased by about 2,000 per c.mm. The mononuclear reaction did not occur until the fifth and following days; they reached a maximum of 11,000 per c.mm. on the sixth day and gradually fell on succeeding days.

Ovarian cells.—Rabbit F. Chart 9.—The cells of two ovaries of a rabbit were injected intraperitoneally, but unfortunately in the middle of the reaction the animal aborted and no further counts were made. There was, however, a marked increase in the mononuclear leucocytes on the third day, the number having risen from 4,000 to 8,250 per c.mm. The polynuclear leucocytes up to this time were gradually diminishing in numbers.

Renal cells.—Rabbit G. Chart 10.—Two reactions closely following one another will be seen on this chart. In the first experiment the dissociated cells of two kidneys of a rabbit were injected intraperitoneally, and in the second experiment the cells of two kidneys of a guinea-pig were similarly injected. In both instances the reaction was again entirely mononuclear, and did not take place until the fourth day after inoculation. The polynuclear leucocytes were not appreciably affected throughout the whole period.

Testicular cells.—Rabbit H. Chart 11.—Intraperitoneal injection of the cells of two testicles of a rabbit again gave a large mononuclear leucocytosis, and the maximum increase was reached on the sixth day. The number of polynuclear leucocytes was diminished during the first few days, but on the fourth day after injection the total number was slightly above that recorded previous to injection.

It will be seen, therefore, that in all these experiments with cellular inoculations there was a notable increase of mononuclear leucocytes four, five or even six days after injection, and no appreciable change in the polynuclear leucocytes throughout the

reactions. An exception may perhaps be taken to the first experiment with Rabbit D., but although in the increase of mononuclear forms on the fifth day there was no rise above the number recorded before inoculation, the number of mononuclear leucocytes had been low for four previous days.

These results agree closely with the observations made by Bullock on the effect of injections of ox blood corpuscles. In his first experiment he injected 13 c.c. intraperitoneally and observed a mononuclear leucocytosis from 3,250 to 9,500 per c.mm. on the fifth and sixth days, and a similar increase of mononuclear leucocytes on the sixth day, with an intraperitoneal injection of 18 c.c. of the same material. There was also an increase of polynuclear leucocytes on the seventh day, but the number of these was high before inoculation.

Bullock found that there was an enormous rise in the immune body at the height of the increase of mononuclear leucocytes, and that the latter followed the fluctuations of the former with regularity.

The results recorded above appear to justify the conclusion that the removal of foreign animal cells is exclusively carried out by mononuclear leucocytes.

Dead tubercle bacillus.—Rabbit E. Chart 8.—The intraperitoneal injection of 10 c.c. emulsion in distilled water of dead tubercle bacillus gave a large oscillating polynuclear reaction. The great increase of polynuclear leucocytes was noted on the fifth day after injection, viz.—14,400 per c.mm., but on the next day the number had fallen to 6,500. The number of mononuclear leucocytes was highest on the third day (10,000 per c.mm.) but it remained somewhat high until the eighth day, the number of polynuclear leucocytes having already fallen to normal for two days.

Extracted tubercle bacillus.—Rabbit G. Chart 10.—Intraperitoneal injection of 10 c.c. of extracted tubercle bacilli produced a slight increase in both kinds of leucocytes, which was greatest on the fifth day. The whole reaction was, however, small, and was probably influenced to a considerable extent by abortion on the second day after inoculation.

Rabbit G. Chart 2.—5 c.c. of extracted tubercle bacilli were injected subcutaneously. There was a late leucocytosis affecting both varieties. The increase commenced on the third day after inoculation, and reached its maximum on the fifth day. A slight diminution of total numbers was observed in the early stages. An interval of a week separated the two reactions.

Tubercle bacillus extract.—Rabbit A. Chart 2.—10 c.c. of tubercle bacillus extract injected intraperitoneally produced an increase in both varieties. The mononuclear leucocytosis was slightly larger, and was maintained for three days, while the polynuclear reaction was noted for two days. The increase, however, of each kind was only slight. On the sixth day after inoculation, this rabbit was again injected intraperitoneally with 15 c.c. of tubercle bacillus extract. Neither kind was much affected for two days, but on the third day, there was an increase per c.mm. of 3,500 mononuclear leucocytes, and 2,500 polynuclear leucocytes. The number had fallen to normal on the next day.

Rabbit G. Chart 10.—Injected with 10 c.c. tubercle bacillus extract intraperitoneally. This injection gave a pure mononuclear reaction, the mononuclear leucocytes gradually increasing until the fifth day, when the number per c.mm. was 8,000.

Rabbit F. Chart 16.—10 c.c. of tubercle bacillus extract were injected subcutaneously. Here again the main effect was on the mononuclear leucocytes, their number increasing from 6,750 to 11,250 per c.mm. on the first day after inoculation. This increase was maintained for three days. The number of polynuclear leucocytes rose slightly (2,500) on the first day after injection but otherwise were scarcely affected.

The injected material in rabbit A was a first extract of tubercle bacilli and may have contained some adherent chemiotactic substances besides the tubercle fatty acid. As a mixed reaction, though mainly affecting the mononuclear leucocytes, was obtained with this injection, the extract was further purified, and in rabbits G and F as above a pure mononuclear leucocytosis was obtained, this extract apparently consisting of pure fatty acid. It may be inferred from these results that the injection of pure tubercle bacilli fatty acid causes a pure mononuclear leucocytosis. The

inoculation of extracted tubercle bacilli gave a reaction of both varieties, but the injected material was not free from wax, having still some acid-fast reaction remaining.

The injection of unaltered tubercle bacilli produced a reaction of both varieties.

The bearing of these observations will be considered later.

Sheep's serum—Rabbit C. Chart 4.—10 c.c. of sheep's serum injected intraperitoneally caused a great rise in number of the mononuclear leucocytes, there being a gradual increase from 4,000 per c.mm. from the day after injection until the fourth day, when there were present 15,000 per c.mm. On the sixth day the number was 6,500 per c.mm. The polynuclear leucocytes were not affected until the day when the mononuclear forms reached their maximum increase, they then rose to 8,000, this being 6,000 more per c.mm. than on the previous day.

The number of both kinds had fallen on the sixth day, and as there was no great difference in the leucocyte count on the eighth day, 20 c.c. of sheep's serum were injected intraperitoneally. Twenty hours afterwards the polynuclear leucocytes were found to be present in very small numbers, only 2,250 per c.mm. The mononuclear leucocytes had increased from 6,000 to 15,000 per c.mm. At the time of taking the blood for this enumeration the animal was very lethargic and blood was only obtained with difficulty, as the peripheral circulation was failing. Death supervened five hours later, and a post-mortem examination revealed the presence of septic peritonitis.

The failure of the polynuclear leucocytes to react will, in the light of other experiments with living bacterial cultures, possibly explain why death resulted so rapidly.

Human pleural effusions and human serum.—Rabbit E. Chart 7.—This animal was being immunized by Dr. Walker for the purposes of other investigations, and I was fortunately able to make blood-counts after several of the injections. The immunizing effect of the latter is well seen in the charts, the reactions gradually decreasing in relation to the total number of leucocytes.

10 c.c. of human pleural fluid injected intraperitoneally produced a gradual increase of both kinds of leucocyte (but more especially the mononuclear form) up to the third day, the numbers then being—polynuclear leucocytes 10,000 per c.mm., mononuclear leucocytes 14,500 per c.mm. There was a gradual fall onwards until the seventh day, when another injection of 15 c.c. of pleural fluid was given. The polynuclear leucocytes were not increased during this reaction, but the mononuclear forms, after falling to 2,000 on the day after injection, rose to 8,500 per c.mm. on the third day after the second injection.

On the fourteenth day 20 c.c. were again injected. A slight hypoleucocytosis was noticed, both kinds being subsequently increased, the mononuclear leucocytes on the third and fourth days after injection, and the polynuclear leucocytes on the third day. Both varieties had fallen to normal on the fifth day after the third injection.

The same animal was injected intraperitoneally after a two months' interval with 20 c.c. of human serum from a case of uræmia. There was a small decrease in both forms for two days and a subsequent slight hyperleucocytosis, involving both mononuclear and polynuclear leucocytes. The return to a normal condition was gradual.

The reaction to pleural fluid, therefore, appears to affect both polynuclear leucocytes and mononuclear forms, but more especially the latter, while serum in uræmia, presumably containing abnormal substances, affected polynuclear leucocytes quite as much as the mononuclear leucocytes.

STAPHYLOCOCCUS PYOGENES AUREUS.

1. *Dead cultures*.—Rabbit F. Chart 9. —One broth culture forty-eight hours old of dead staphylococcus pyogenes aureus was injected intraperitoneally. This reaction was remarkable for the large mononuclear leucocytosis on the fifth day after inoculation—an increase from 7,250 to 18,600 per c.mm. The number of mononuclear leucocytes for the first few days was slightly diminished. The number of polynuclear leucocytes increased a little on the first day, but diminished for the next

three days. The largest total recorded was on the fifth day, namely, 6,400 per c.mm.

Rabbit K. Chart 14.—10 c.c. of a forty-eight hours old broth culture of dead staphylococcus pyogenes aureus, produced an increase mainly in the mononuclear leucocytes, these rising from 4,000 to 12,000 approximately on the second day. The return to normal occupied three days. There was a slight increase of polynuclear leucocytes (1,750) on the first day, but a subsequent fall during four days to the low total of 1,750.

Rabbit Q. Chart 14.—There was again a marked increase of mononuclear leucocytes following the injection of one dead agar culture of staphylococcus pyogenes aureus forty-eight hours old, especially on the third day, when the number reached 18,400 per c.mm. This increase was only noted on one day. The polynuclear leucocytosis was larger than in the previous experiment. The highest number recorded was 12,800 per c.mm. but on the next day, they had fallen to 8,500, and were still further diminished on subsequent days. It will thus be seen that injections of dead staphylococci produce by far the greatest effect on the mononuclear leucocytes, and generally late in the reaction. There was practically no increase of polynuclear leucocytes in two of the experiments, and in rabbit Q. the polynuclear leucocytosis was not very large.

2. *Living cultures.*—Rabbit T. Chart 14.—Half an agar culture forty-eight hours old of living staphylococcus pyogenes aureus produced an extremely large polynuclear leucocytosis. On the third day after inoculation, the polynuclear leucocytes numbered 24,500 per c.mm., and on the fifth day, 14,000. The mononuclear leucocytes also increased to some extent on both these days, but the number never exceeded 11,000 per c.mm. (7,000 before inoculation).

With the same injection, there was a similar reaction in Rabbit R., Chart 14, allowing for individual susceptibility. The polynuclear leucocytes increased in number from 6,000 to 10,900 per c.mm. on the second day. On the same day, there was an increase of 2,000 mononuclear leucocytes, and this was maintained when the number of polynuclear leucocytes had fallen to normal again.

These two reactions to living cultures form a marked contrast to those in which dead cultures were inoculated.

Everard, Demoor and Massart, found after injecting 1 c.c. of a culture of living *staphylococcus pyogenes aureus* intramuscularly a polynuclear leucocytosis which was present until the seventy-second hour. Up to this time the mononuclear leucocytes had not increased in number.

Muir obtained similar results with injections of 1 to 2 c.c. of pure cultures of *staphylococcus pyogenes aureus* having a low virulence. A considerable interval, however, elapsed between the leucocyte counts in some of his observations.

These observations show that, while the reaction to dead staphylococci, which had been subjected to a temperature of 100°, was entirely comparable to the reactions to indifferent animal cells, the reactions to cultures containing living staphylococci and their unaltered products affects the polynuclear leucocytes most markedly. The latter, therefore, may probably be regarded as engaged in the destruction of the micro-organisms and their toxins, while the former act in relation to the dead cocci precisely as they do with other cells.

BACILLUS TYPHOIDÆ ABDOMINALÆ.

Rabbit D. Chart 5.—Subcutaneous injection of 0.5 c.c. of a broth culture twenty-four hours old of living *bacillus typhoidæ abdominalæ* produced a large polynuclear leucocytosis on the third and fourth days. The number of mononuclear leucocytes was high (18,000) before injection, but after falling for the first few days after inoculation they increased to 12,000 per c.mm.

Seventeen days after this injection the animal was inoculated intraperitoneally with two agar cultures, forty-eight hours old, of living *bacillus typhoidæ abdominalæ*. There was an immediate rise of polynuclear leucocytes the following day from 2,000 to 8,000, and this was maintained on the second day after injection. During the next three days the number of leucocytes (polynuclear) was normal.

The mononuclear leucocytes were only slightly increased for four days after injection, but on the fifth day suddenly rose to 8,000 per c.mm., twice the number they were before inoculation.

After nine weeks' interval one forty-eight hour old agar culture of living bacillus typhoidæ abdominalæ was again injected, the animal's blood having been counted for a week previously. On the day after injection (about 20 hours) there was an enormous rise of mononuclear leucocytes, consisting chiefly of lymphocytes, from 4,250 to 19,750 per c.mm., and an increase in polynuclear leucocytes from 3,000 to 11,750 per c.mm. At the same time the animal was extremely ill and collapsed (10 a.m.) but its condition gradually improved during the day. The number of mononuclear leucocytes had fallen to 7,000 on the second day, but that of the polynuclear leucocytes increased still further to 16,660 per c.mm., the latter gradually diminished on the three following days, but the number of mononuclear leucocytes during the later stages of the reaction was higher than usual, although only slightly so.

Compare this early and sudden mononuclear leucocytosis associated with extreme intoxication, with the similar increase after injection of infected serum on Chart 4.

Rabbit H. Chart 11.—One agar culture three days old of living bacillus typhoidæ abdominalæ produced the greatest change in the mononuclear leucocytes during the first few days of the reaction, the number rising on the second day from 3,000 to 11,800 per c.mm., and being maintained with slight variations for four more days. The number of polynuclear leucocytes rose on the second day from 4,500 to 7,400 per c.mm., but it was not until the fifth and sixth days that they were greatly increased.

The early mononuclear leucocytosis was very similar in the last two experiments, but in rabbit H the polynuclear increase was late, not being fully established until the sixth day. The latter animal, however, was not nearly so seriously ill as rabbit D on the day after injection.

PNEUMOCOCCUS.

Rabbit N. Chart 6.—Subcutaneous injection of $8\frac{1}{2}$ c.c. of broth culture forty-eight hours old of dead pneumococcus. In this

experiment, there was an increase of polynuclear leucocytes from 3,660 to 10,000 per c.mm., a mononuclear leucocytosis from 8,160 to 15,160 per c.mm. on the day after injection, and a subsequent rise on the fourth and eighth days of each kind of leucocyte. It will be seen from the chart that the increase of each variety was coincident in point of time.

Rabbit F. Chart 16.—The reaction to 0·5 c.c. broth culture two days old of living pneumococcus injected subcutaneously was only slight. Both kinds were increased, but especially the polynuclear leucocytes, which reached a maximum of 7,000 per c.mm. on the second day after injection; the mononuclear leucocytes attained their greatest number (10,400) on the fourth day.

Schlesinger's experiments.—Subcutaneous injection of ·05 c.c. of a pneumococcus culture gave a gradual increase of polynuclear leucocytes from 4,000 to 9,500 per c.mm. till the ninth hour, while the lymphocytes gradually decreased from 8,000 to 3,000 per c.mm. until the seventh hour, and remained the same until death, which occurred thirteen hours after inoculation.

A ·15 c.c. broth culture of pneumococcus (diplococcus of Fraenkel) producing death on the following day, gave an increase of 1,000 polynuclear leucocytes on the day of injection, after which both varieties gradually diminished till death. Just before death, the numbers of polynuclear and mononuclear leucocytes were 1,000 and 1,750 per c.mm. respectively.

Sterile broth.—This was given to show the effect of the fluid mediums in which some of the cultures were injected.

Rabbit F. Chart 9.—10 c.c. injected intraperitoneally produced a moderate polynuclear leucocytosis on the following day, the number increasing from 3,200 to 10,500 per c.mm. The mononuclear leucocytes also increased in number from 3,500 to 5,500 during the same time, and there was a further increase of mononuclear leucocytes on the third day, the number then being 7,250 per c.mm.

Rabbit H. Chart 11.—20 c.c. injected intraperitoneally gave a small leucocytosis of both kinds. This was only maintained for one day.

"PEPTONE."

Rabbit O. Chart 13.—1 grm. of peptone in 3 c.c. normal salt solution was injected intraperitoneally. On the following day the number of both kinds of leucocyte was diminished. There was a progressive increase of the polynuclear forms until the fourth day, when the number was 12,250. The number of mononuclear leucocytes remained low until the fourth day, when there was an increase from 4,250 to 9,750 per c.mm. Both varieties had fallen to normal on the fifth day after inoculation.

DIPHTHERITIC TOXIN.

Rabbits M. and L. Chart 12, were each injected subcutaneously with .02 c.c. and rabbit A. with 0.2 c.c. of very powerful diphtheritic toxin. In all cases there was an enormous polynuclear leucocytosis.

Rabbit M. died during the evening of the first day after inoculation. The number of polynuclear leucocytes rose from 4,000 to 17,000, and the mononuclear leucocytes diminished from 6,750 to 3,500 per c.mm.

Rabbit A. died during the evening of the second day after injection. On the first day, the number of polynuclear leucocytes increased from 4,500 to 9,500 per c.mm., the mononuclear forms were slightly diminished. On the morning of the second day, there was a leucocytosis of polynuclear leucocytes of 17,000, and of mononuclear leucocytes of 14,500 c.mm.

Rabbit L. also died on the second day after inoculation, at 2.25 p.m. On the first day, the number of polynuclear leucocytes rose from 3,250 to 12,000, the mononuclear leucocytes remained unaltered. At 10 a.m. on the second day, the polynuclear forms had still further increased to 18,500, and the mononuclear leucocytes had risen to 12,000 per c.mm. In the blood-count one hour before death, the number of polynuclear forms had fallen to 5,000, and that of the mononuclear variety to 5,800 per c.mm.

In the fourth experiment with rabbit J. a much smaller dose (.005 c.c.) was injected subcutaneously with the view of determining, if possible, the behaviour of the leucocytes over a longer

period. There was again a very large and early polynuclear leucocytosis, the numbers increasing on the second day from 6,000 to 20,500. The mononuclear leucocytes did not increase appreciably until the third day, when the number rose from 7,250 to 13,250 per c.mm. The number of polynuclear forms was falling at this time. There was a leucocytosis of both varieties for three more days, and on the seventh day after injection, the mononuclear leucocytes suddenly increased from 11,500 to over 22,000 per c.mm. Corresponding with the large mononuclear increase, there was a slight rise of polynuclear leucocytes. Both kinds had fallen to 8,000 on the ninth day after injection.

Rabbit O. Chart 13, was injected with an even smaller dose of toxin, viz., .001 c.c. The reaction was, however, very similar to the previous experiment with rabbit J.

The polynuclear leucocytes were the first to be affected, the number reaching a maximum of 10,750 on the second day, and then gradually falling to normal. The mononuclear leucocytes were not materially altered during the first three days, but on the fourth day, the number rose to 10,330 per c.mm., and on the fifth day was 8,750.

These experiments agree with Besredka's results, in that the injection of the diphtheritic toxin produces a large and rapid polynuclear leucocytosis, but they also show that the mononuclear leucocytes play an important part, and react at a later period. In four out of the five experiments, there was a marked increase of this kind on the third or fourth day, and in rabbit J. a very large mononuclear leucocytosis on the seventh day after inoculation. No doubt a large number of polynuclear leucocytes are destroyed in the process of neutralizing the toxin, and possibly the phagocytic action of the mononuclear leucocytes may be called into play in removing these, as the late mononuclear reaction is very similar to that following injections of the various cells which have been previously described.

It will also be noticed that there is again a sudden preagonistic rise of mononuclear leucocytes, as in the injection of infected sheep's serum and bacillus typhoidæ abdominalæ.

MICROCOCOCCUS RHEUMATICUS (living).

Rabbit O. Chart 13, was intravenously injected with five blood-agar cultures forty-eight hours old of very attenuated living micrococcus rheumaticus. The number of polynuclear leucocytes increased on the first day after inoculation from 2,500 to 10,500 but fell the next day to 6,000; on the fourth day again the number per c.mm. was 9,000. The mononuclear leucocytes reached a maximum on the fourth day of 8,750, this being an increase of 3,750 per c.mm.; for the first three days they remained unaltered.

Rabbit V. Chart 15.—A similar reaction was produced by the subcutaneous injection of three agar cultures forty-eight hours old. A polynuclear leucocytosis was established on the first day after inoculation and was maintained for four days. The mononuclear leucocytes were unaltered for the first three days but their number increased on the fourth day from 6,500 to 9,500 per c.mm.

Rabbit U. Chart 15.—Three agar cultures forty-eight hours old were injected subcutaneously. A polynuclear leucocytosis was observed on the next day and was maintained more or less for two days. The mononuclear leucocytes reacted much earlier than in rabbit V. The number increased on the first day from 4,500 to 8,500 per c.mm. and this increase was very nearly maintained for three days. These reactions again show the early polynuclear reaction to the living organism, and the late mononuclear reaction possibly to remove the dead material.

Schlesinger's experiments.—1 c.c. of a broth culture of streptococcus erysipelatus injected subcutaneously produced a polynuclear leucocytosis which was highest on the fifth day, the number of leucocytes increasing from 4,000 to 17,000 per c.mm. The mononuclear forms reached a maximum increase on the ninth day of 4,800, the total number then being 11,800 per c.mm.

The number of both kinds of leucocyte was falling at the maximum of infiltration.

BACILLUS DIPHTHERIÆ AMERICANÆ (living).

The toxin of this organism is very highly toxic, but the organism itself is extremely inactive, large doses failing to kill a guinea-pig.

Rabbit P. Chart 15.—0.25 c.c. of an agar culture forty-eight hours old of the living organism injected subcutaneously produced a fall in polynuclear leucocytes for two days, and a gradual increase from the third until the fifth day, but at this time the number was only very slightly above that recorded previous to inoculation. The number of mononuclear leucocytes increased on the third day from 4,000 to 8,750 and remained so until the next day. On the 6th day the number had fallen to 4,250 per c.mm.

Rabbit S. Chart 15.—One agar culture seventy-two hours old injected subcutaneously gave a leucocytosis on the third day almost entirely confined to the polynuclear leucocytes; the mononuclear leucocytes were not appreciably altered.

The two reactions are not comparable to any extent. The dose injected was, however, very much larger in rabbit S, and it is possible, with an interval of twenty-four hours between each leucocyte count, that the polynuclear forms were at one time higher than appears in the instances recorded.

Schlesinger's experiments.—Injection of broth culture (8 c.c.) of bacillus diphtheriæ Americanæ. The number of polynuclear leucocytes increased at the fourth hour from 3,800 to 6,500 per c.mm., but fell at the sixth hour to about 2,000 and remained at 3,000 till death on the following morning. The mononuclear leucocytes numbered 10,500 per c.mm. before injection. They diminished in the early stages of the reaction, but one hour before death the number was 9,000 per c.mm.

.15 c.c. of a broth culture of bacillus diphtheriæ Americanæ killed with .5 per cent. phenol gave a rise and fall on succeeding days affecting both varieties, but mainly the polynuclear leucocytes. The polynuclear leucocytes numbered 25,000 and the mononuclear leucocytes 11,000 per c.mm.

4 c.c. of virulent diphtheritic culture injected subcutaneously caused a leucopenia during the first few hours, and a subsequent

increase in both kinds of leucocyte. Previous to death the polynuclear leucocytes numbered 14,000 (an increase of 9,000) and the mononuclear kind 22,000 (an increase of 17,000).

CONCLUSIONS.

1. The injection in rabbits of dead non-toxic animal matter, such as animal cells and red blood corpuscles, produces a reaction exclusively affecting the mononuclear leucocytes. Inoculation with pure fatty acid from the tubercle bacillus gives a similar reaction. (*Cf.* the alleged mononuclear leucocytosis—lymphocytosis—of unmixed tuberculosis).

2. The injection of toxins produces a pure polynuclear leucocytosis, unless death ensues rapidly. In the later stages, as the number of polynuclear leucocytes diminish, the mononuclear leucocytes increase in number, presumably to remove the excess of dead material.

3. The reaction to serum which contains chiefly indifferent animal matters, is mainly mononuclear.

4. Micro-organisms subjected to heat, whereby bacterium and toxin are destroyed, produce on injection the greatest effect on the mononuclear leucocytes.

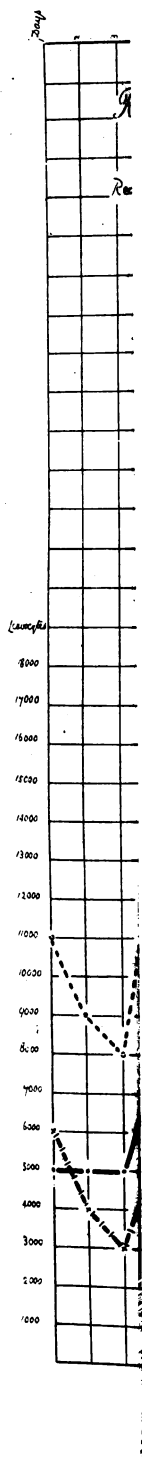
5. The injection of living micro-organisms produces mainly a polynuclear leucocytosis, or a mixed leucocytosis in which the mononuclear reaction is secondary.

Much more work must necessarily be done in this direction before any definite conclusions can be drawn, but these experiments, I think, bear out the suggestion that in rabbits, the polynuclear leucocytes are chiefly responsible for the destruction of toxic products and living bacteria, while the mononuclear leucocytes deal chiefly with non-toxic matter and nutritive substances.

Finally, I have to thank Dr. Ainley Walker very sincerely, not only for the preparation and inoculation of all cultures, etc., but for his kindly encouragement and guidance throughout the research.

REFERENCES.

- Muir. ... "The bone-marrow and leucocyte production." *Journ. of Path.*, 1901.
- Bullock. ... "On the nature of hæmolytic and its relation to bacteriolysis." *Trans. Path. Soc. Lond.*, 1901, p. 208.
- Everard. ... "Sur les modifications des leucocytes." *Annales de l'Institute Pasteur*, 1893.
- Demoor,
Massart.
- Haushalter. *Compt. Rend. Soc. de Biol. Paris*, 1899, p. 678.
- Spillman.
- Roger. ... *Compt. Rend. Soc. de Biol. Paris*, 1899, p. 436.
- Josué.
- Ehrlich. ... "Die Anämie." *Histology of Blood. Trans. by Myers*, 1900.
- Schlesinger. *Zeitschrift. f. Hyg. u. Infect. Krank. Leipzig*, 1900, p. 349.
- Milroy. ... *Jour. of Physiol.*, 1899. Vol. 25, p. 105.
- Malcolm.
- Brinkerhoff, *Jour. of Med. Research. Boston*, March, 1902, p. 173.
- Tyzzar.
- Bruce. ... *Proc. Roy. Soc.* Vol. 55, p. 295.
- Werigo. ... "Les globules blancs comme protecteurs du sang." *Annales de l'Institute Pasteur*, 1892. Vol. 6, p. 478.
- Durham ... *Journ. of Pathol.* Vol. 6, 1897, p. 338.
- Sherrington. *Proc. Roy. Soc.*, 1894. Vol. 55, page 161.
- Romer. ... *Berlin Klin. Woch.*, 1891. No. 36.
- Batty-Shaw. *Journ. of Pathol.*, March, 1902. Vol. 8, p. 70.
- Besredka. ... "De la leucocytose dans la diphthérie." *Annales de l'Institute Pasteur*, 1898. T. 12.
- Achard ... *Compt. Rend. Soc. de Biol. Paris*, 1901, 52, pp. 486, 487.
- Lœper.
- Ramsden. ... *Journ. of Physiol.* Vol. 28, July, 1902. *Proc.*, p. 23.
- Hankin ... *Proc. Camb. Philosoph. Soc.*, Feb., 1892.
- Kanthack.
- Nicolas, ... "Étude sur la leucocytose dans l'intoxication par la toxine diphthérique." *Archiv. de Med. Experiment*, 1897.
- Courmont.
- Goldscheider, "Variationen der Leucocytose." *Zeitsch. für Klin. Med.*, 1894. Bd. 25, p. 373.
- Jacob.
- Lowit. ... "Studien zur Physiol. und Pathol. des Blutes und der Lymph." *Yena*, 1892.
- Ibid.* ... "Über die Beziehungen der Leucocyten zur bactericiden Wirkung des Blutes." *Ziegler's Beiträge zur Pathol. Anat.*, 1897. Bd. 24.
- Havet. ... "Du rapport entre le pouvoir bactericide du sang du chien et sa richesse en leucocytes." *La Cellule*, 1894, T. 10.



Days

Rabbit A 15 days
Rabbit G

Reactions to: 1 7

2.

3.

Leucocytes

14000

13000

12000

11000

10000

9000

8000

7000

6000

5000

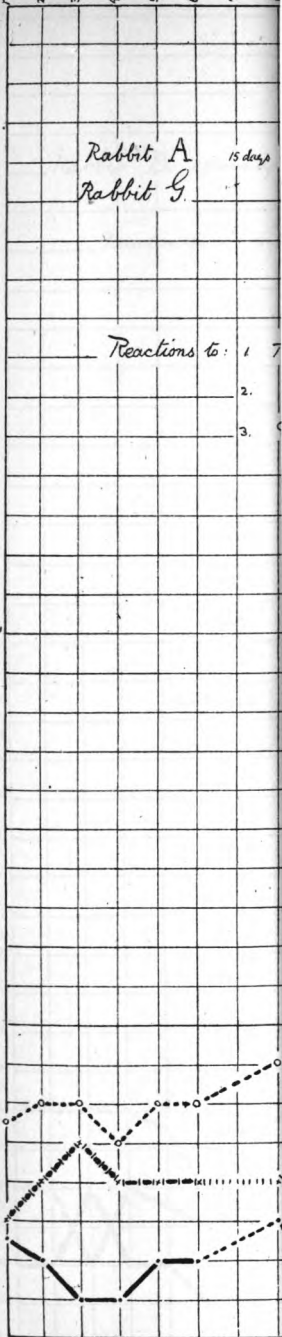
4000

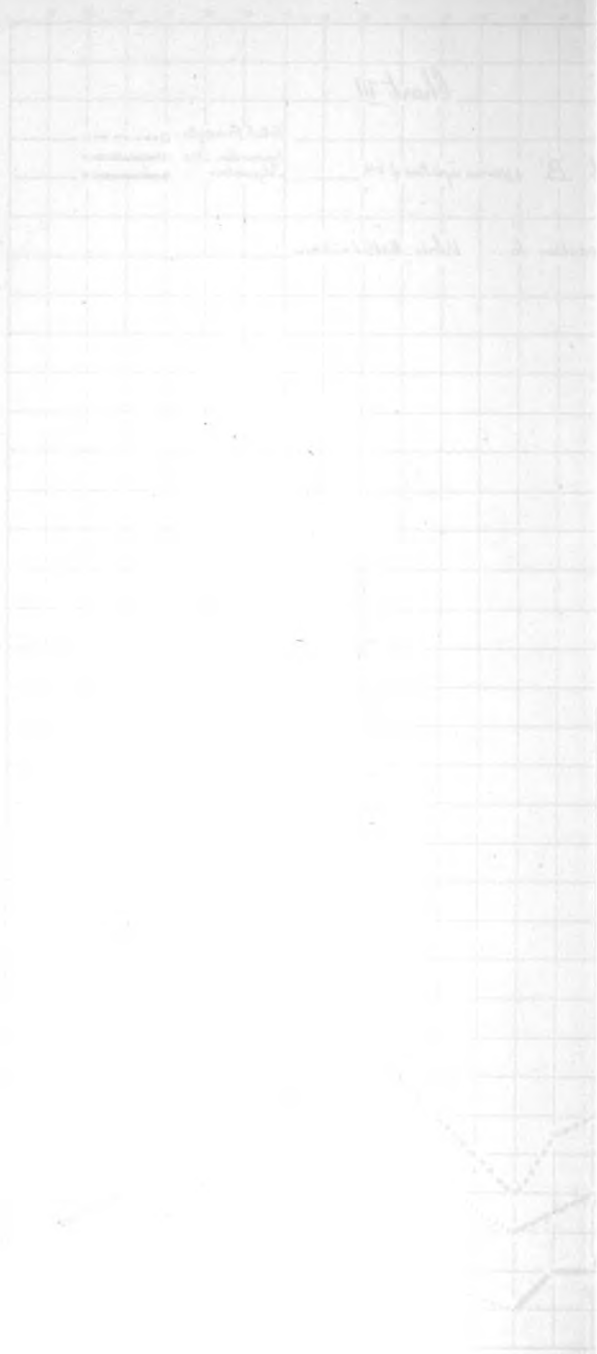
3000

2000

1000

0





Rabbit C.			
Reaction to Sheep's Serum.			

Polynuclear

× 1000 × 1000 × 1000 ×

● **RESEARCH** ●



10cc. Sheep's Serum intraperitoneally

20 c.c. Sheep's Serum intraperitoneally

Death 5 hrs later from septic peritonitis.



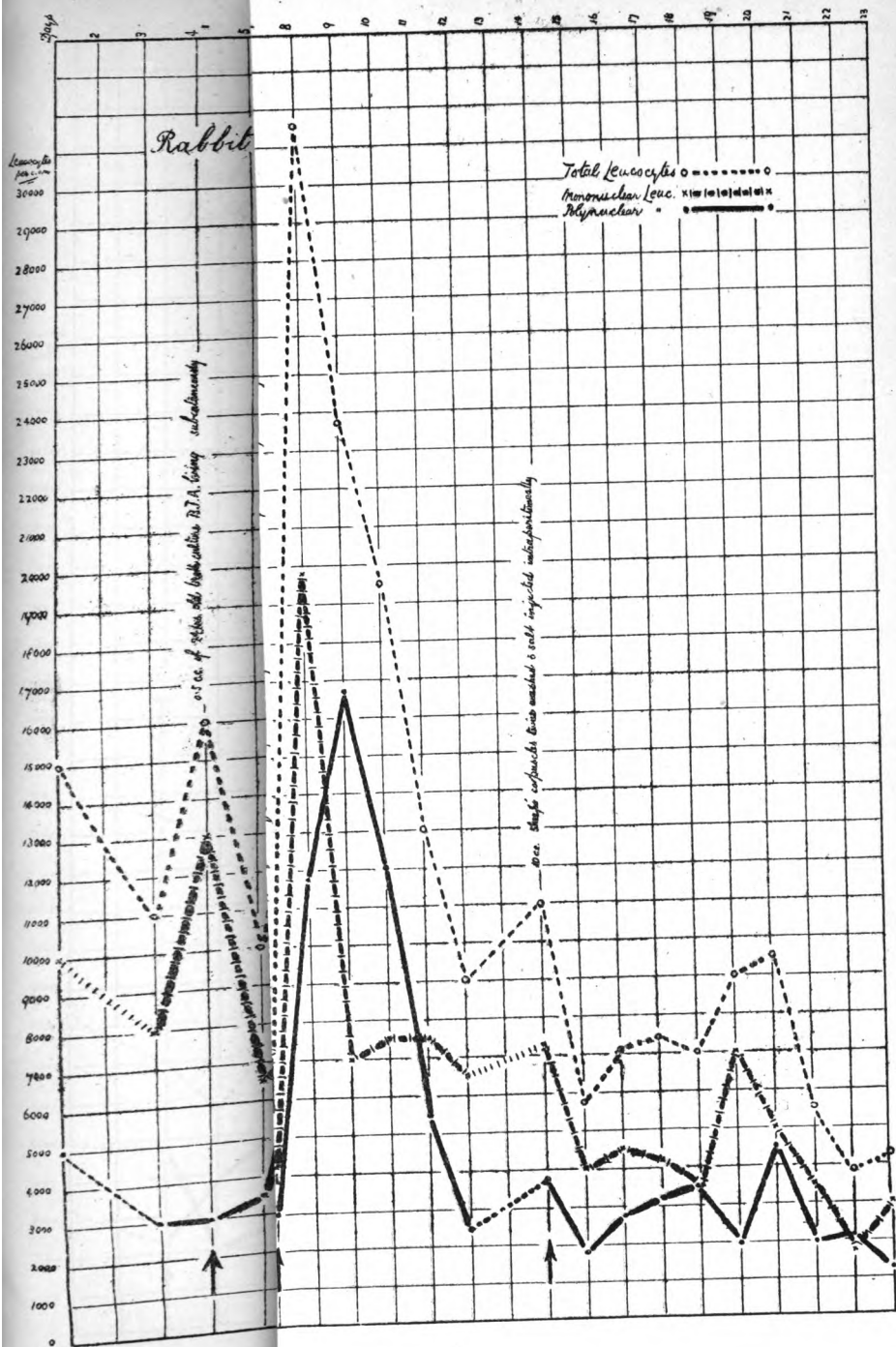
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Diagram or drawing area with faint grid lines and some faint lines.

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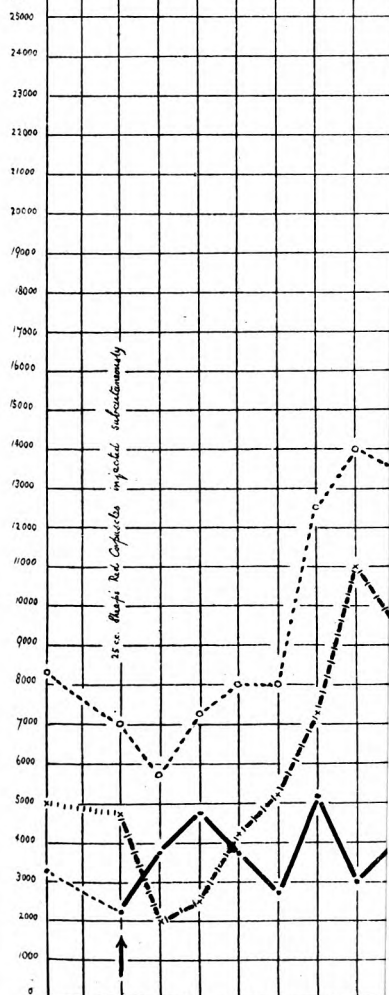


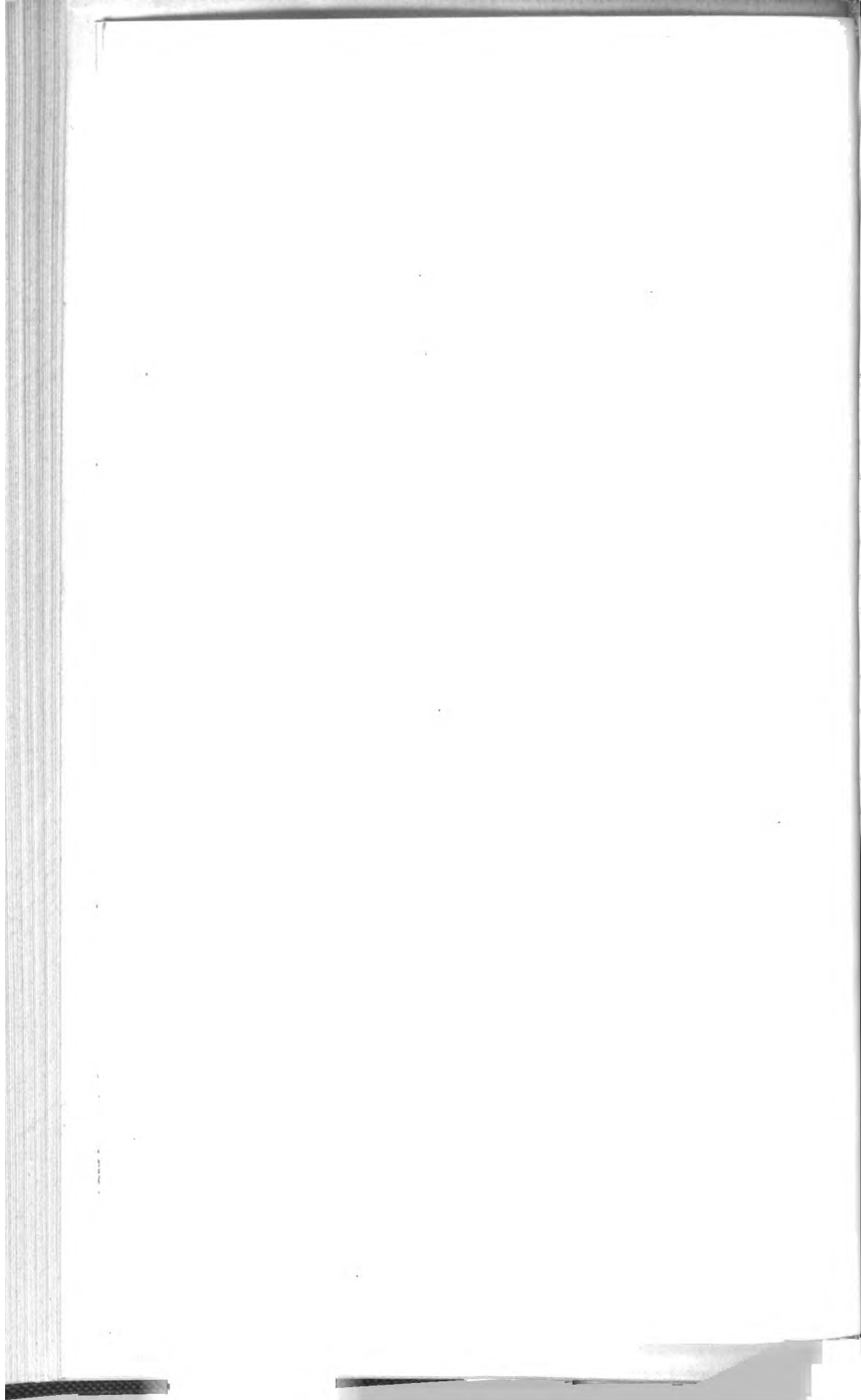


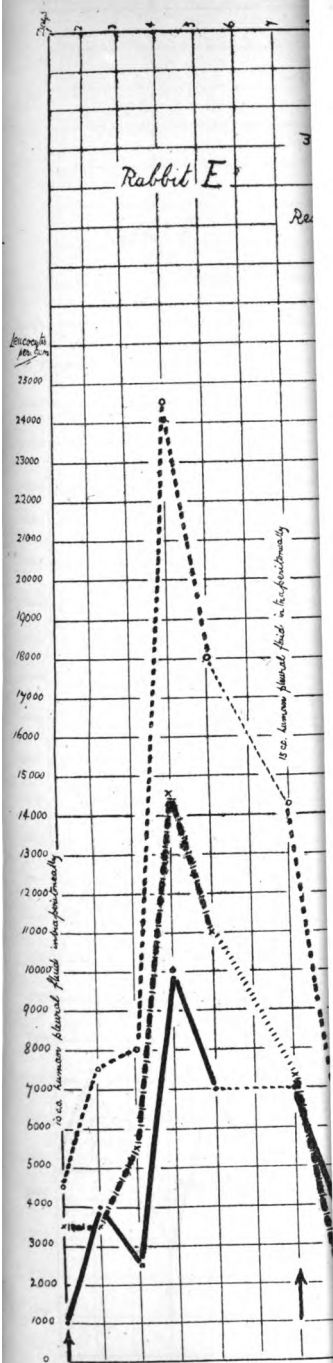
Days

Rabbit D. with reaction to
Rabbit N. with reaction to

Leucocyte
count







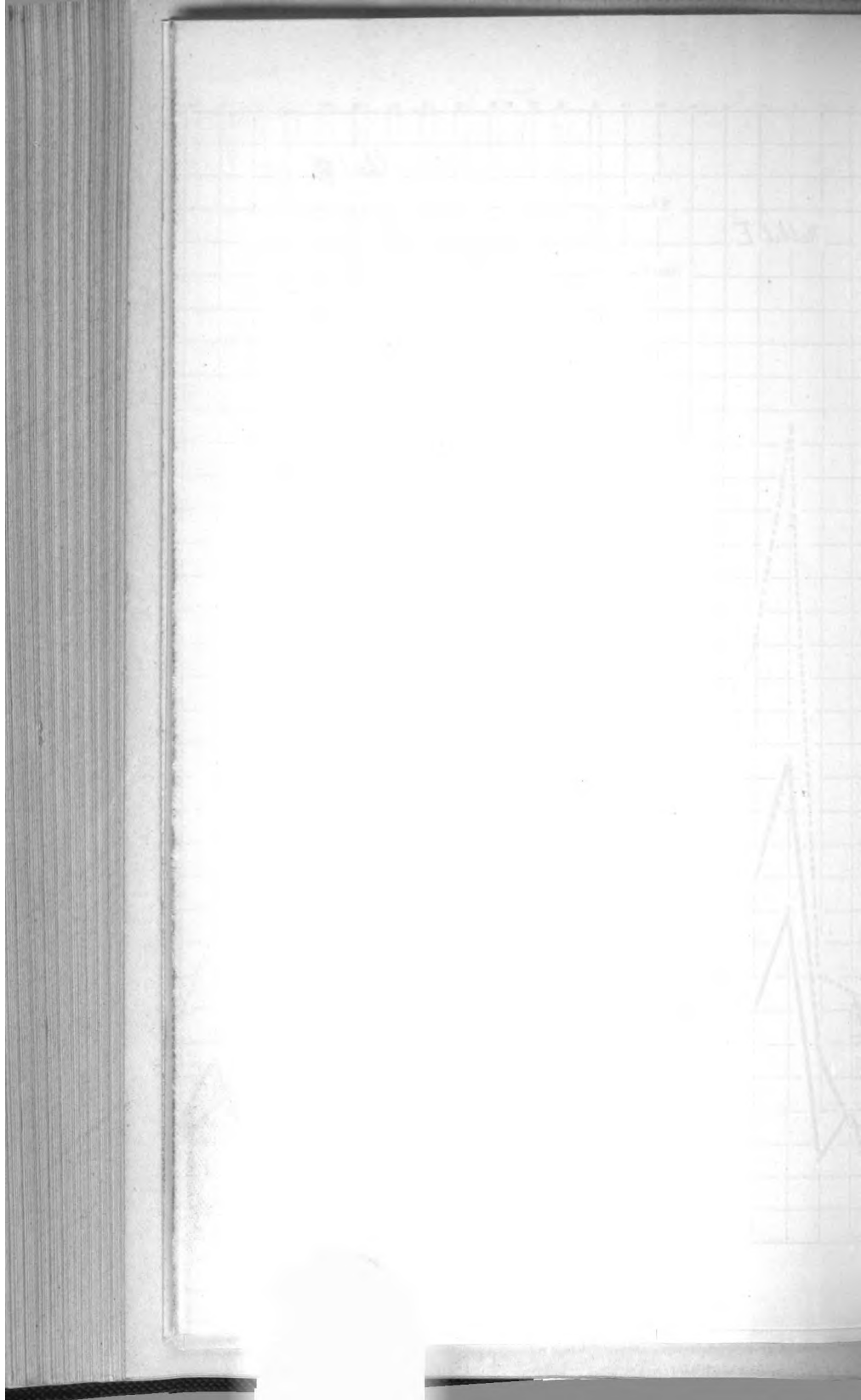
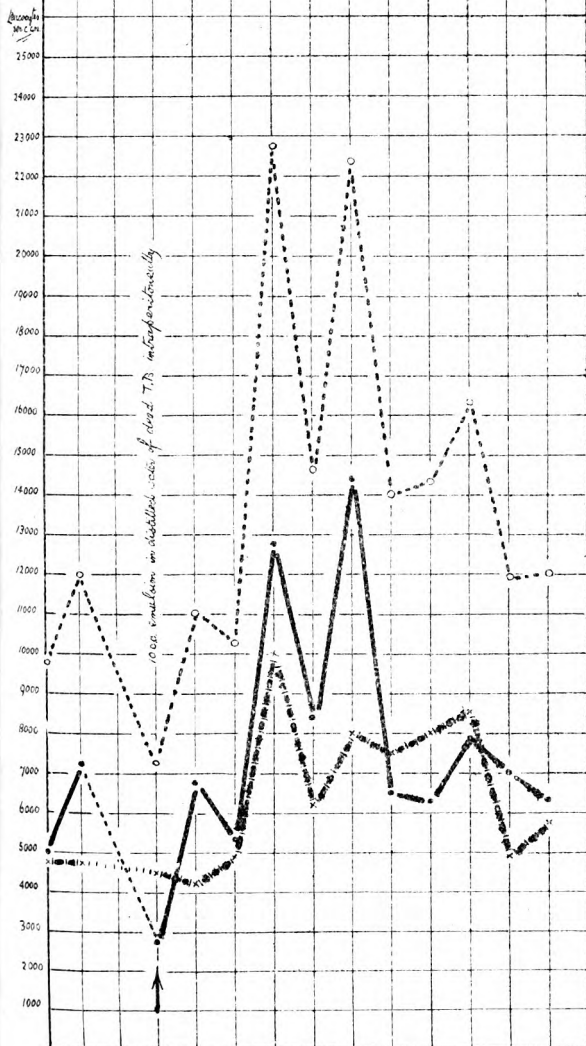


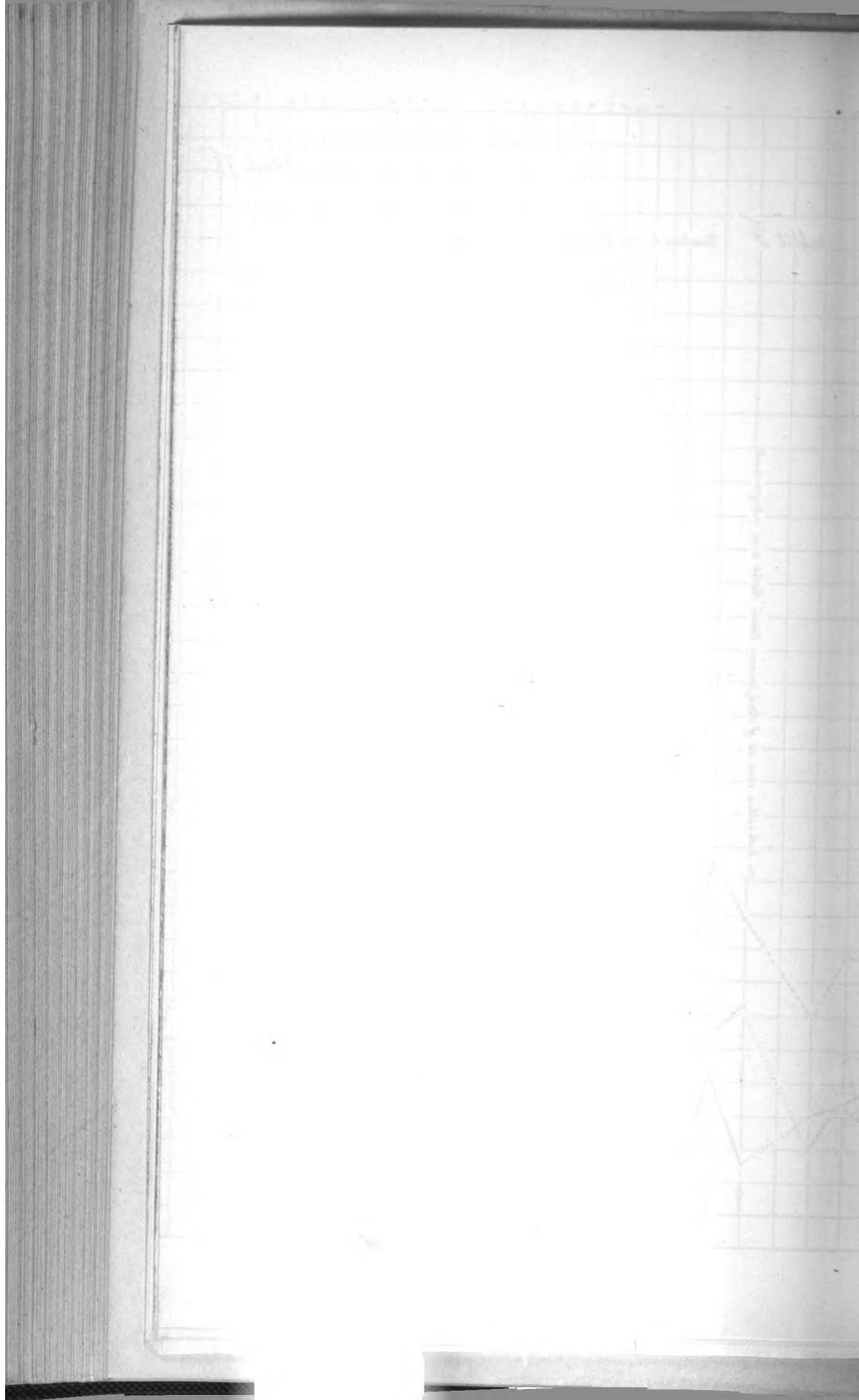
Chart VIII

Rabbit E

Reaction to Tubercle Bacillus (dead)

Total Leucocytes
Mononuclear / sec.
Polynuclear





Amount
for Gen

12 000

11 000

10 000

9 000

8 000

7 000

6 000

5 000

4 000

3 000

2 000

1 000

0

0

0

0

0

0

0

0

0

0

0

0

0

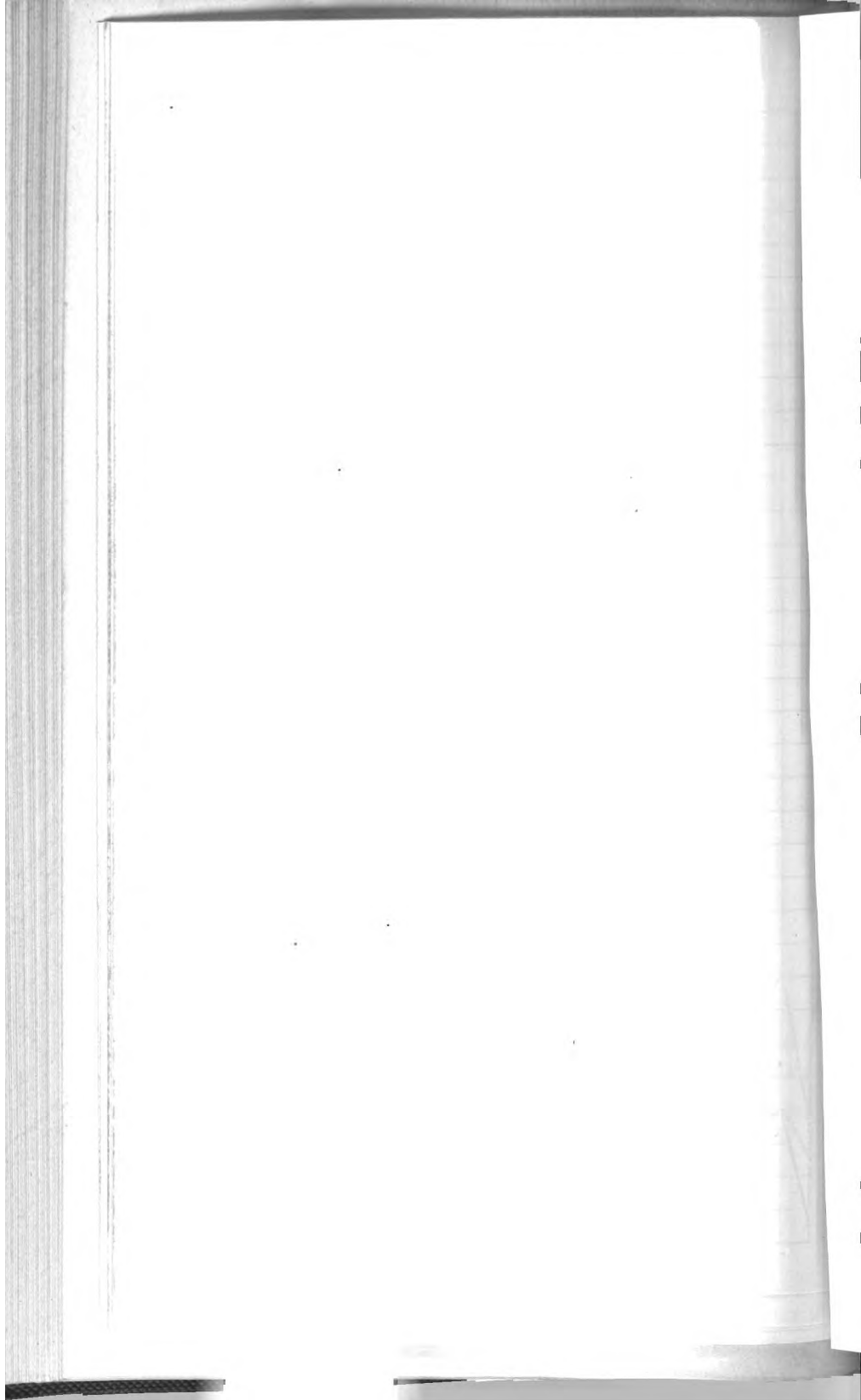
0

0

0

0

0



100000
100000

25000

24000

23000

22000

21000

20000

19000

18000

17000

16000

15000

14000

13000

12000

11000

10000

9000

8000

7000

6000

5000

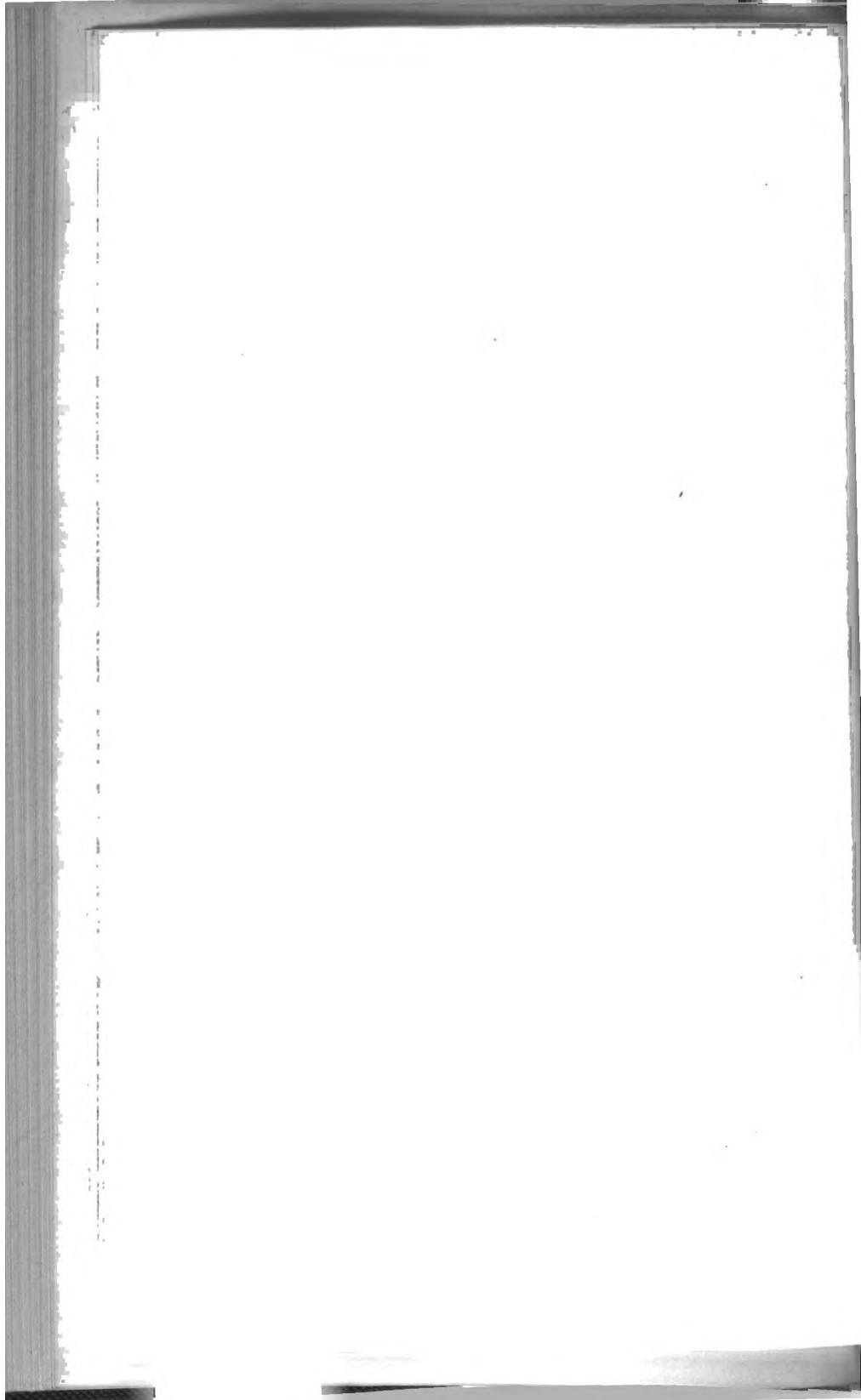
4000

3000

2000

1000

0



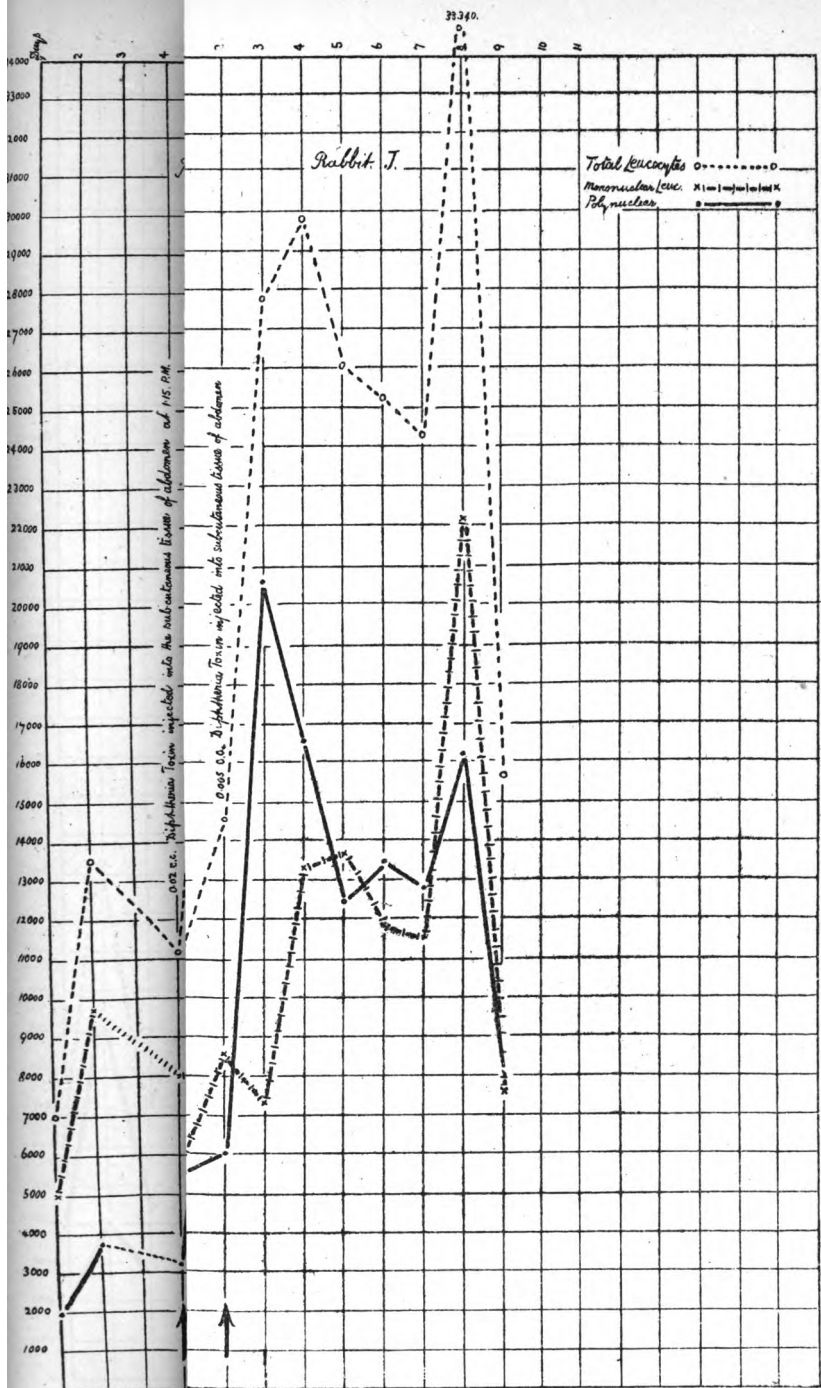


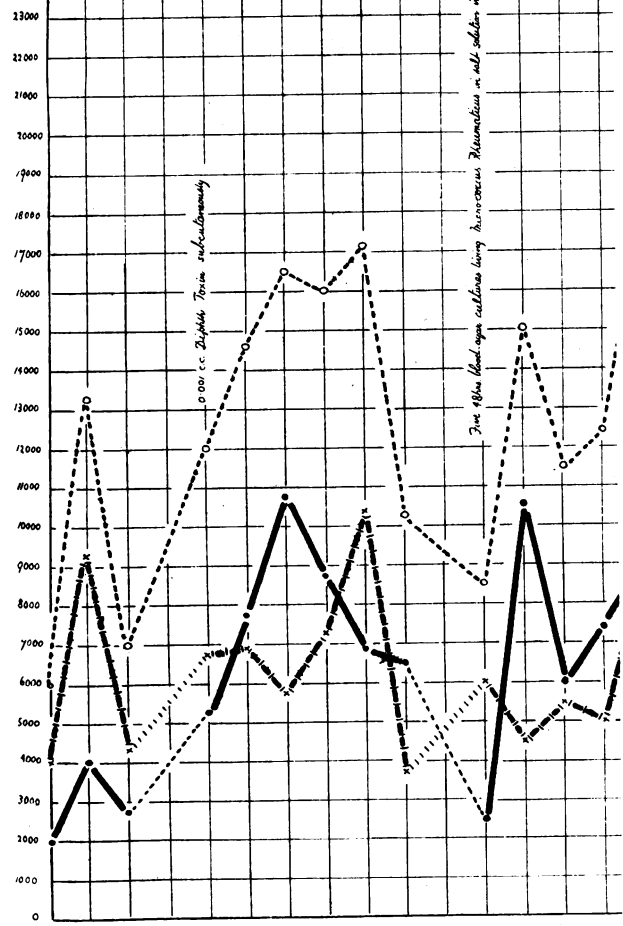


Chart XIII

Rabbit O.

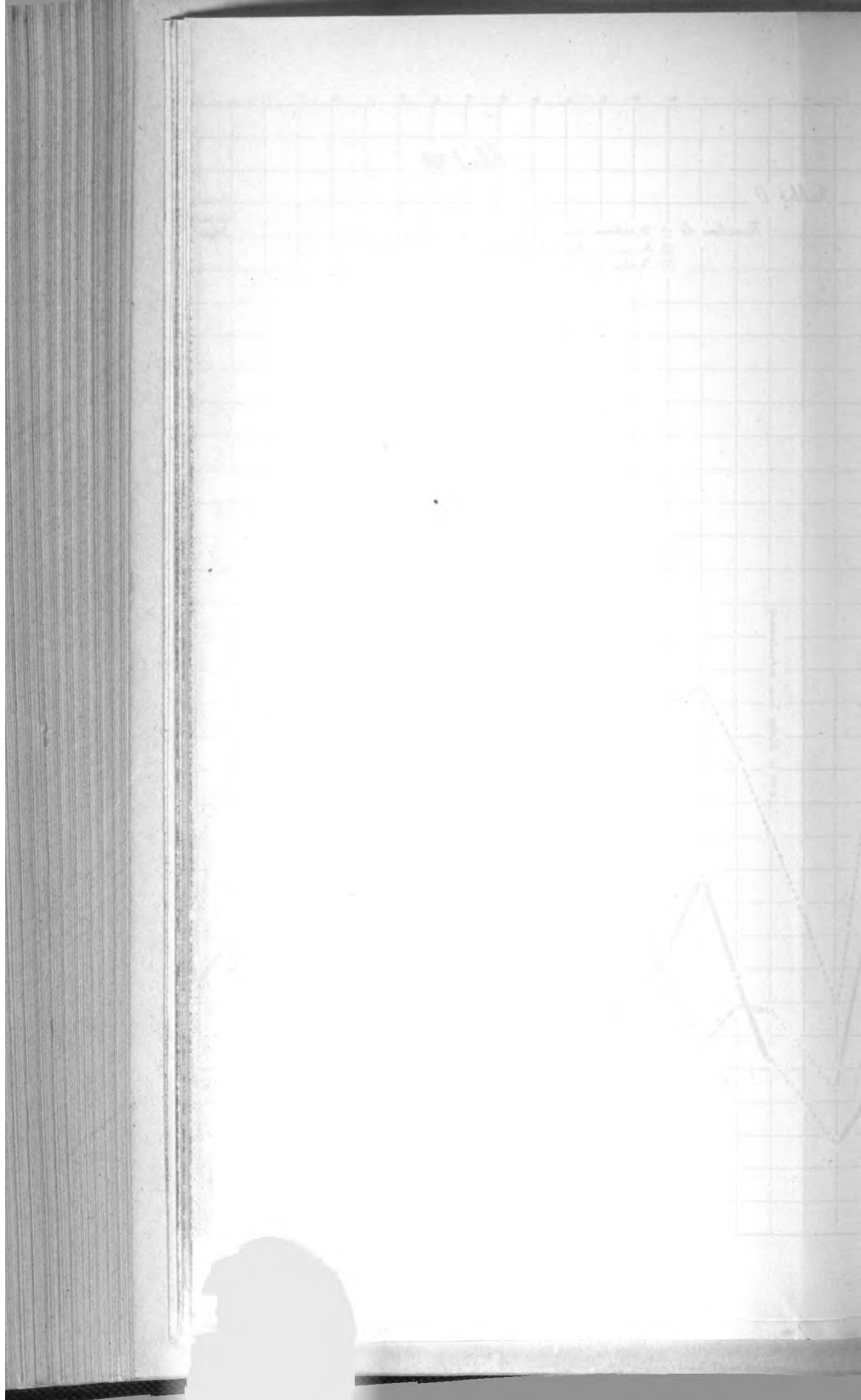
- Reactions to
- ① Diphtheria Toxin
 - ② Micrococcus Rheumaticus (living)
 - ③ Krypton

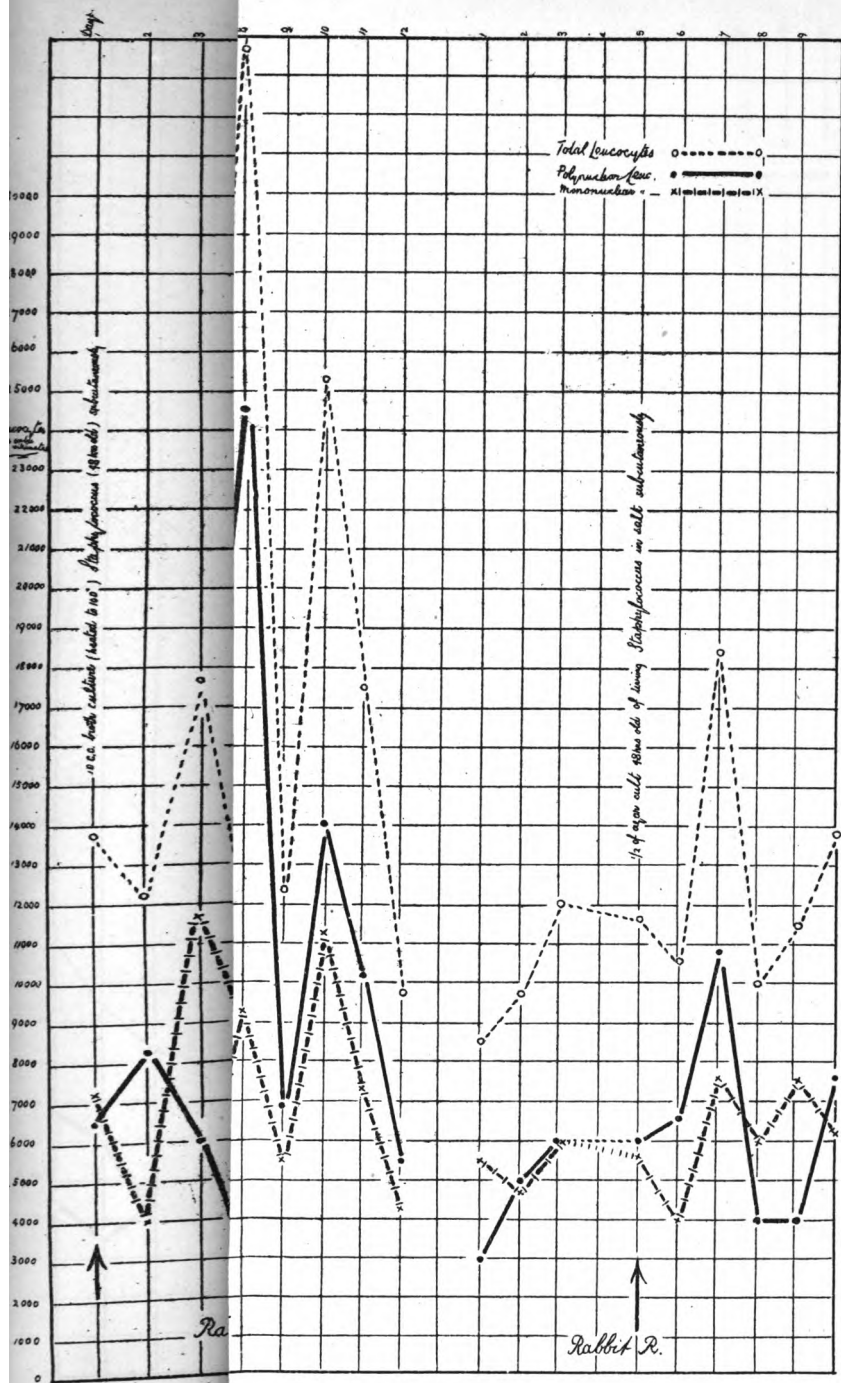
Reaction
per c.c.

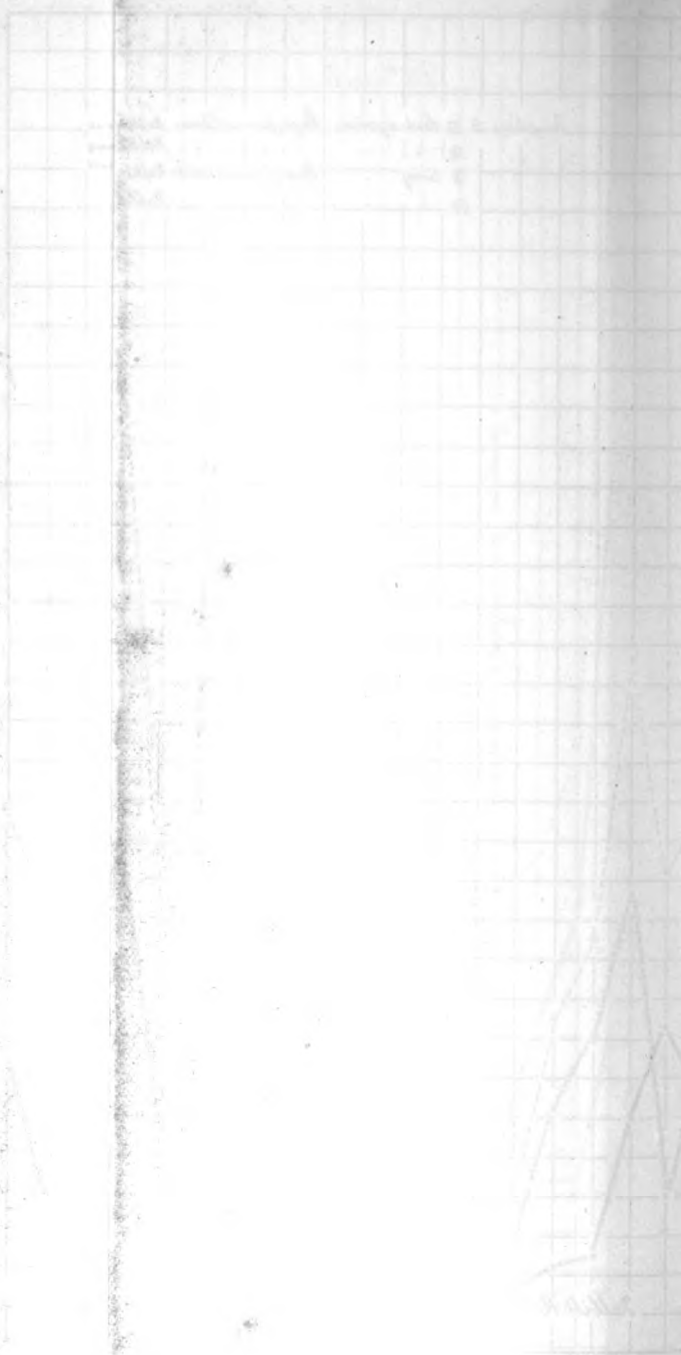


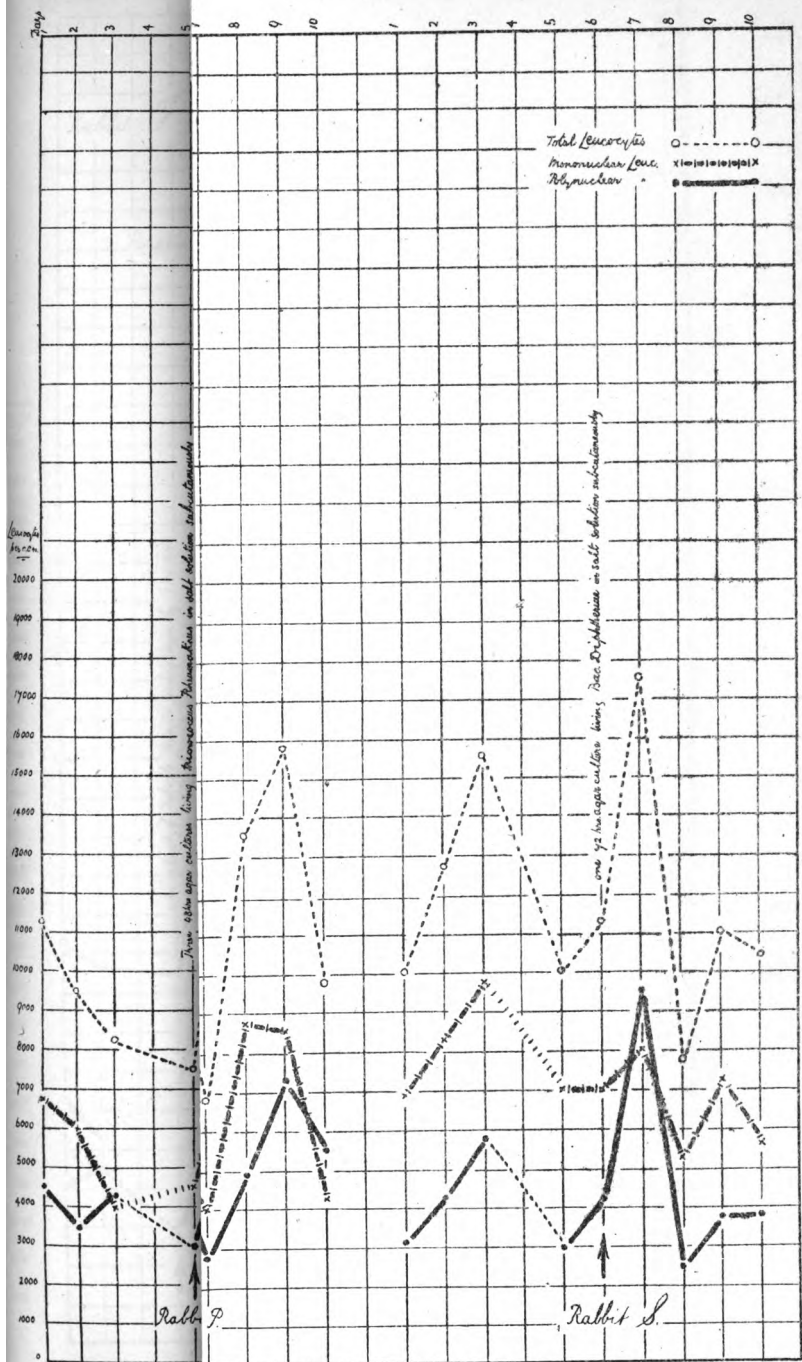
0.001 c.c. Diphtheria Toxin subcutaneously

Two 0.5 cc blood agar cultures living Micrococcus Rheumaticus in left subcutaneous (3 cc fluid aspirate)









Days

Rabbit F.

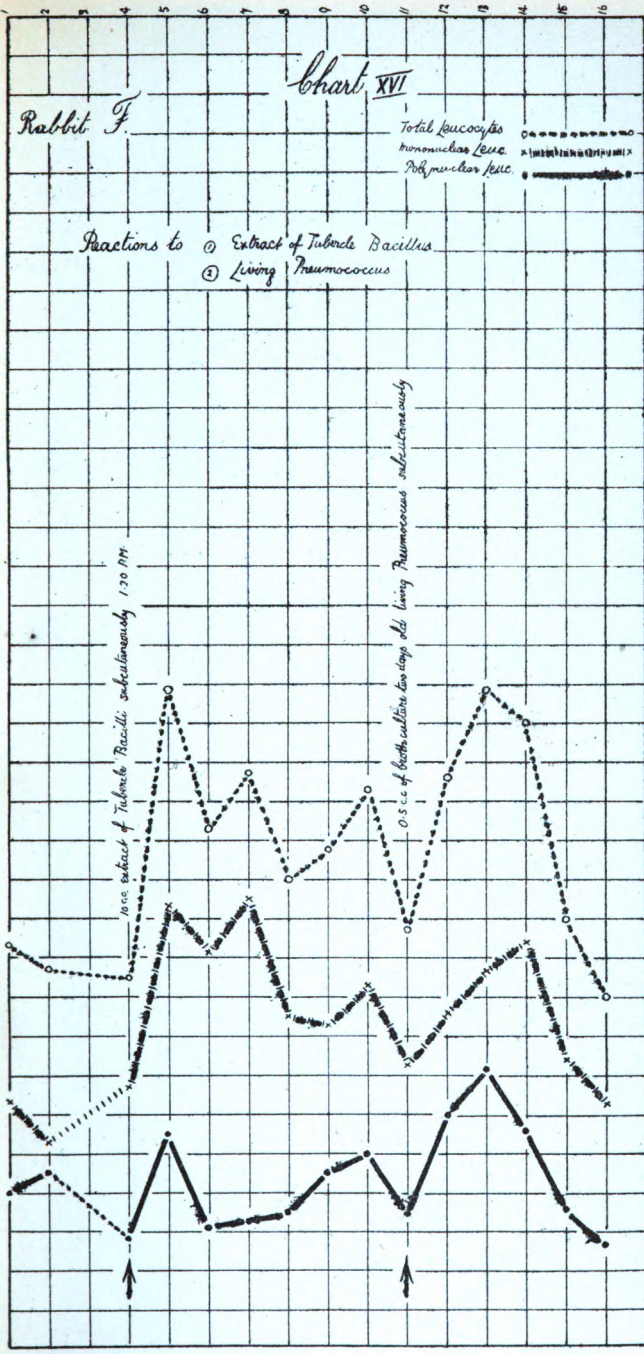
Chart XVI

Total leucocytes
mononuclear leuc.
polynuclear leuc.

Reactions to ① Extract of Tubercle Bacillus
② Living *Typhimurium*

Leucocytes per c.c.

23000
22000
21000
20000
19000
18000
17000
16000
15000
14000
13000
12000
11000
10000
9000
8000
7000
6000
5000
4000
3000
2000
1000
0





TABLES.

The weight is given in grammes.

The temperature was taken on all occasions in the rectum.

The figures of all corpuscles (red and white) represent the number per c.mm.

RABBIT (1).

Day.	Weight.	Temp.	Total Leu.	
1	2130	100	7250	
2	2130	102.2	7500	
3	2170	100.6	8500	
4	2190	101.4	7500	
5	2175	100.2	—	
6	2120	103	—	
7	2205	102	7000	
8	2180	100.2	6000	
9	2120	102.2	7000	
10	2120	102.6	6000	Injection of pneumococcus. ? Dose.
11	2050	104.4	3000	
12	1990	103.8	8000	
13	2010	104.9	8500	
14	1935	104.6	3500	
15	1895	102.4	6000	
16	1840	102.8	12000	
17	1810	100.4	10000	
18	1910	101	12000	
19	1880	102.4	10500	
20	1945	101.2	9500	
21	1910	101	9000	
22	1960	101.2	9500	

RABBIT (2).

Day.	Weight.	Temp.	Total Leu.	
1	2165	101	9500	
2	2170	100.8	8000	3 c.c. T. B. extract, sub- cutaneously.
3	2250	101	11000	
4	2265	99.8	6500	10 c.c. T. B. extract, intra- peritoneally.
5	2320	102	18500	
6	2290	103	15000	
7	2360	102.2	10000	
8	2350	102	9000	
9	2365	102.4	8500	
12 days interval				
22	2435	102	8000	15 c.c. T. B. extract, intra- peritoneally.
23	2425	102.2	8000	
24	2400	103	20500	
25	2335	100.4	21000	
26	2370	101.2	14000	
27	2345	101	29000	
28	2320	101.5	24500	
29	2470	101.8	12500	
30	2435	101.6	10000	

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RABBIT (3).

Day.	Weight.	Temp.	Total Leu.	
1	2240	99.2	10500	
2	—	—	—	
3	—	—	—	
4	2310	101	10000	10 c.c. red corpuscles (sheep), intraperitoneally.
5	2320	102.2	11500	
6	2280	101	10500	
7	2240	100.2	13500	
8	2310	101	8000	
9	2280	100.6	8500	15 c.c. red corpuscles (sheep), intraperitoneally.
10	2300	101	8000	
	7 days interval			
18	2360	102.4	14500	
19	2410	100.2	10500	
20	2440	100.4	12000	
21	2475	99.8	10000	
22	2460	99.6	11500	10 c.c. red corpuscles, intra- peritoneally.
23	2425	103.2	45000	
24	2335	102.5	10500	
25	2370	102	18000	
26	2350	102	24000	
27	2325	102.8	11500	
28	2295	101.2	31500	
29	2255	100.2	21500	
30	2210	100.8	17000	
31	—	—	—	
32	2330	100.6	14500	
33	2280	100.2	12500	
34	2245	101	13500	

RABBIT (4).

February 24th, 1902, 5 c.c. human serum intraperitoneally.
March 2nd, 1902:—

Day.	Weight.	Temp.	Total Leu.	
1	1885	101.2	9000	
2	1900	101	8500	10 c.c. human serum, intra- peritoneally.
3	1900	101.7	11000	
4	1825	100.2	14000	
5	1800	99	13500	
6	1765	100.2	11500	
7	1700	102.2	8500	
	8 days interval			
16	2105	101	16500	
17	2050	100.2	17000	10 c.c. human pleural fluid, intraperitoneally.
18	1995	102.6	19000	
19	1895	103	30500	
20	1765	100.8	20500	
21	1745	100.6	19500	
22	—	—	—	
23	1790	100.2	17000	
24	1740	100.2	13000	

RABBIT "A".

Day.	Weight.	Temp.	Total Leu.	
1	1585	102.4	9500	
2	1560	101.2	9000	
3	—	—	—	
4	—	—	—	
5	1660	101.4	10500	
6	1695	100	9000	
7	1630	103.6	9000	
8	1690	101.7	9000	0.5 c.c. broth culture, 24 hours old V.M. heated to 80° C., subcutaneously.
9	1605	103.2	10000	
10	1595	102.2	8500	
11	—	—	—	
12	1750	101.2	8000	1 c.c. broth culture, living V.M. 24 hours old, subcutaneously.
13	1825	102.2	8500	
14	1860	102.4	12000	
15	1840	102.4	8500	5 c.c. broth culture, living V.M. 24 hours old, subcutaneously.
16	1715	105	2000	
17	1700	103.4	25500	
18	1825	102.8	11500	
19	1780	103	10000	
20	1850	102.2	9000	
21	1885	102.4	8500	3 agar cultures, 24 hours old living V.M., subcutaneously
22	1735	104	5000	
23	1765	103	20500	
24	1778	103	13500	
25	1780	102.6	7500	
33	1720	7 days interval 101.6	8000	3 c.c. broth culture, 24 hours old living B.T.A., subcutaneously.
34	1705	103.4	12500	
35	1685	102.6	10000	
36	1670	102.2	7500	
37	1620	102.4	9500	
38	1630	102	8500	
46	1970	7 days interval 101.8	10000	
47	1965	101.6	9000	5 c.c. broth culture, 24 hours old living B.T.A., subcutaneously.
48	1895	103	17000	
49	1885	102.6	22250	
50	1835	102.2	11500	
51	1810	102.4	15500	
52	—	—	—	
53	1910	101.6	14000	
54	1845	102	9000	
55	1790	101.2	10500	
56	—	—	—	3 agar cultures, 48 hours old living V.M., subcutaneously

RABBIT "A"—continued.

8 days interval from last count.

Day.	Weight.	Temp.	Total Leu.	Poly. Leu.	Mon. Leu.	
1	—	—	11000	5000	6000	
2	—	—	9000	5000	4000	
3	—	—	8000	5000	3000	
4	—	—	14000	8000	6000	
5	—	—	11000	7000	4000	
6	—	—	—	—	—	
7	—	—	—	—	—	2 agar cultures, 72 hours old living V. M. in broth, subcutaneously
8	2030	102	14000	8000	6000	
9	1985	104·8	8000	5000	3000	
10	1915	103·4	18000	11000	7000	
11	1885	103	15000	10000	5000	
12	1860	102·6	12000	9500	2500	
13	—	—	—	—	—	
14	—	—	—	—	—	
15	—	—	—	—	—	
16	1990	102·6	14000	8000	6000	
17	1970	102·8	10000	4000	6000	
18	1965	102·6	9000	3000	6000	
19	1935	103·4	3000	1000	2000	
20	1905	102·8	4000	2000	2000	
21	—	—	—	—	—	
22	1930	102	8000	4000	4000	3 agar cultures, 24 hours old living V. M. in broth, subcutaneously
23	2025	103	9000	4000	5000	
24	2000	103·4	3000	1000	2000	
25	1965	103·4	3000	2000	1000	
26	1930	104	5000	2000	3000	
27	1885	103·4	6000	4000	2000	
28	—	—	—	—	—	
29	2030	104·2	7000	5000	2000	
30	1960	103·8	16000	10000	6000	
31	2000	105·2	10000	8000	2000	5 c.c. T. B. extract, sub- cutaneously.
32	1985	105	8000	6000	2000	
33	1985	104·8	—	—	—	
34	1965	103·8	16000	6000	10000	
35	—	—	—	—	—	
36	1920	103·6	10000	4000	6000	
37	1840	103	—	—	—	
38	1930	103·8	9000	6000	3000	
39	1875	103	9000	5000	4000	
40	1830	102·2	12000	7000	5000	
41	1800	103	9000	5000	4000	

RABBIT "A"—continued.

15 days interval from last count.

Day.	Weight.	Temp.	Total Leu.	Poly. Leu.	Mon. Leu.	
1	2100	103·6	5500	2500	3000	
2	2090	103·4	6000	2000	4000	
3	2050	102·2	6000	1000	5000	
4	—	—	5000	1000	4000	
5	2120	102·9	6000	2000	4000	
6	2160	103·6	6000	2000	4000	
7	—	—	—	—	—	
8	2195	103·2	7000	3000	4000	
9	2090	102·8	7000	2000	5000	10 c.c. T.B. extract, intra-peritoneally.
10	2020	102·6	7000	4000	3000	
11	1960	103	10500	4000	6500	
12	1935	102·4	8500	2000	6500	
13	2020	102·8	8000	2000	6000	
14	—	—	—	—	—	
15	2230	103·8	9000	4000	5000	15 c.c. T.B. extract, intra-peritoneally.
16	2130	103·6	8000	2500	5500	
17	2090	103·4	8500	4000	4500	
18	2040	103	15000	6500	8500	
19	2122	103·4	8000	3500	4500	
3½ months interval						
1	2920	103	10800	5200	5600	
2	2870	102·7	9250	4500	4750	0·2 c.c. diphtheria toxin, subcutaneously.

RABBIT "A"—continued.

Day.	Weight.	Temp.	Total Leu.	Poly. Leu.	Mon. Leu.	Large Mono.	Large Lymph.	Small Lymph.
3	2750	105	14000	9500	4500	135	585	3780
4	2760	98	31500	17000	14500	290	5220	8990

This rabbit died during the evening of the next day, 8 to 12 hours after last count.

RABBIT "B."

Day.	Weight.	Temp.	Total Leu.	Poly. Leu.	Mon. Leu.	
1	2240	100	7500			
2	2215	102	7000			
3	2350	100	11000			
4	2145	100·6	15000			
5	2210	100·6	9500			
6	2150	102·2	9000			
7	2190	102	8000			
8	2250	101·6	7000			
9	2200	101·6	7000	—	—	1 c.c. broth culture, 24 hours old living V.M., intraperitoneally.
10	2190	102·3	3000			
11	2150	102·6	7500			
12	2140	103·2	7000			
13	2205	102·6	8000			
14	—	—	—			
15	2205	102·6	9000	—	—	5 c.c. broth cultures, 24 hours old living V.M., intraperitoneally.
16	2050	102·2	14000			
17	2030	102·2	13000			
18	1990	100·8	9000			
19	2040	100	9500			
20	2020	102·2	8000			
8 days interval						
29	2210	101·5	7000			
30	2190	101	9000			
31	2200	103	6000	—	—	1 agar culture, 24 hours old living V.M., intraperitoneally.
32	2190	102·6	20000			
33	2120	102·4	6000			
34	2170	102·6	17000			
35	2215	102·4	15000			
46	—	—	—	—	—	2 agar cultures, 48 hours old living V.M., subcutaneously.
3 months interval						
1	2300	103·2	5500	2000	3500	
2	2310	102·8	6000	2000	4000	
3	2230	102	6000	4000	2000	
4	2235	102·6	6000	2000	4000	
5	2260	103·1	5500	2000	3500	
6	2125	103·2	4000	1000	3000	
7	—	—	—	—	—	
8	—	—	—	—	—	
9	2225	103	7000	2000	5000	
10	2165	102·6	8500	2000	6500	1 agar culture, 24 day old living V.M., subcutaneously.
11	2125	103	5500	2000	3500	
12	2005	102·6	14000	9000	5000	
13	1965	101·6	8000	4000	4000	
14	1995	102·6	7000	2000	5000	
15	—	—	—	—	—	
16	2240	102·8	9000	4000	5000	
17	2160	104·4	7000	3500	3500	
18	2125	103·6	7000	3000	4000	

RABBIT "C."

Day.	Weight.	Temp.	Total Leu.	Poly. Leu.	Mon. Leu.	
1	3220	102	9500			
2	3190	102	14500			
3	3025	102·2	9500			
4	2985	102·2	18000			
5	2965	102·2	6500			
6	2970	102·2	12000			
7	—	—	—			
8	3080	103	7500			
9	3010	102·6	7500	—	—	10 c.c. sheep's serum, intra- peritoneally.
10	3010	102·8	7000			
11	2905	102·4	12500			
12	2860	102·4	15000			
13	2810	103·6	15000			
14	2935	101·6	13000			
15	2850	101	10500			
			8 days interval			
24	2700	100·2	7500			
25	2685	102	18500			
26	2525	100	15000			
27	2495	100	33000			
28	2440	100	29000			
29	—	—	—			
30	2635	102	32000			
31	2570	100	17000			
32	2600	100	11000			
33	2640	100·2	8500			

The observation on the 33rd day was on March 12, 1902.

March 27th, 1902, 10 c.c. sheep's serum, intraperitoneally.

April 9th, " " " "

May 12th, " " " "

October 7th, 1902:—

1	3270	102	7000	2250	4750	
2	3270	102·6	7000	500	6500	
3	3400	102·4	6500	3500	3000	
4	3320	102·8	5330	3330	2000	
5	3370	102·6	12000	6000	6000	
6	—	—	—	—	—	
7	3270	103	9250	3500	5750	10 c.c. sheep's serum, intra- peritoneally.
8	3260	104	7250	3500	3750	
9	3380	103	11660	4000	7660	
10	3390	102·8	12000	2500	9500	
11	3450	102·6	23500	8500	15000	
12	3460	103·8	11000	4500	6500	
13	—	—	—	—	—	
14	3450	102·4	8960	2960	6000	20 c.c. sheep's serum, intra- peritoneally.
15	3270	101·6	17250	2250	15000	

This rabbit died 4 hours after the last count. A post-mortem examination showed the presence of septic peritonitis.

RABBIT "D."

Day.	Weight.	Temp.	Total Leu.	Poly. Leu.	Mon. Leu.	
1	2260	102·4	15000	5000	10000	
2						
3	2195	102·2	11000	3000	8000	
4	2180	102·2	16000	3000	13000	0·5 c.c. 24 hours old broth culture, living B.T.A., subcutaneously.
5	2147	103·4	10000	3500	6500	
6	2055	103	16500	9000	7500	
7	2020	102·8	23000	11000	12000	
8	2070	102	12000	6000	6000	
9	—	—	—	—	—	
10	2298	102	17000	7000	10000	
11	2220	103·4	9000	4000	5000	
12	2190	103	11000	4500	6500	
13	2170	102·4	13000	5000	8000	
14	2225	102·4	8000	2000	6000	
15	2200	—	—	—	—	
16	—	—	—	—	—	2 agar cultures, 48 hours old living B.T.A., intraperitoneally.
17	2320	102·4	6000	2000	4000	
18	2305	104	12500	8000	4500	
19	2300	103·2	11500	8000	3500	
20	2210	102·5	7500	2500	5000	
21	2220	102	7500	2500	5000	
22	2135	102·2	11000	3000	8000	
		10 weeks interval				
1	2500	103·2	7750	4250	3500	
2	2520	102·4	7000	2000	5000	
3	2550	102	12500	5500	7000	
4	2540	102·2	2500	1250	1250	
5	2570	102·2	8000	3500	4500	
6	—	—	—	—	—	1 broth culture, living B.T.A. 48 hours old, intraperitoneally. Chiefly lymphocytes. Very collapsed.
7	2540	102·2	7250	3000	4250	
8	2540	98·8	31500	11750	19750	
9	2450	102·4	23660	16660	7000	
10	2390	103	19500	12000	7500	
11	2380	103·8	13000	5500	7500	
12	2300	103·6	9000	2500	6500	
13	—	—	—	—	—	10 c.c. sheep's red corpuscles, intraperitoneally.
14	2150	103·6	11000	3750	7250	
15	2120	103·4	5750	1750	4000	
16	2080	103·6	7160	2660	4500	
17	2130	103·8	7400	3200	4200	
18	2080	103·2	7000	3500	3500	
19	2080	103	9000	2000	7000	
20	2010	103·4	9500	4500	5000	
21	2060	102·8	5500	2000	3500	
22	2060	103	3800	2160	1640	
23	2000	102·6	4250	1250	3000	

RABBIT "D"—continued.

16 days interval.

Day.	Weight.	Temp.	Total Leu.	Poly. Leu.	Mon. Leu.	
40	2220	—	8160	3160	5000	
41	—	—	—	—	—	25 c.c. sheep's red corpuscles, subcutaneously.
42	2220	103·2	7000	2250	4750	
43	2170	105·2	5750	3750	2000	
44	2120	104·8	7250	4750	2500	
45	2150	104·2	8000	3750	4250	
46	2140	103·6	8000	2750	5250	
47	2220	103·6	12500	5160	7340	
48	—	—	14000	3000	11000	
49	2080	—	13500	4000	9500	
50	2070	—	12000	5000	7000	
51	2070	—	9500	3500	6000	
52	—	—	9000	4000	5000	

RABBIT "E."

June 16th, 1902, 10 c.c. human pleural fluid, intraperitoneally.

June 20th, " " " " "

July 2nd, " " " " "

July 8th, 1902:—

Day.	Weight.	Temp.	Total Leu.	Poly. Leu.	Mon. Leu.	
1	2245	103	4500	1000	3500	10 c.c. human pleural fluid, intraperitoneally.
2	2150	103·4	7500	4000	3500	
3	2020	102·2	8000	2500	5500	
4	1985	101·5	24500	10000	14500	
5	2070	101·6	18000	7000	11000	15 c.c. human pleural fluid, intraperitoneally.
6	—	—	—	—	—	
7	2235	102·6	14330	7000	7330	
8	2120	104·2	6000	4000	2000	
9	2090	102·6	8500	5500	3000	
10	2125	102·2	13800	5300	8500	
11	2215	103	7500	2000	5500	20 c.c. human pleural fluid, intraperitoneally.
12	2220	—	—	—	—	
13	—	—	—	—	—	
14	2335	103·4	10000	4500	5500	
15	2225	104·8	7000	4500	2500	
16	2300	103	7000	3000	4000	
17	2280	103	12000	5500	6500	
18	2270	—	12500	5250	7250	
19	2250	—	6500	3500	3000	

RABBIT "E"—continued.

10 weeks interval.

Day.	Weight.	Temp.	Total Leu.	Poly. Leu.	Mon. Leu.	
1	2570	103	8000	3000	5000	20 c.c.human serum (from uræmia), intraperitoneally.
2	2570	103	9500	5500	4000	
3	2480	103.4	6660	3660	3000	
4	2480	102.6	7000	3500	3500	
5	2540	102.6	8750	5250	3500	
6	—	—	—	—	—	
7	2490	103	9500	5500	4000	
8	2510	105.6	8250	5750	2500	
9	2450	105	6250	4000	2250	
10	2490	103.2	12500	7500	5000	
11	2500	103.2	12160	6160	6000	
12	2490	103	9000	4680	4320	
13	—	—	—	—	—	
14	2550	102.2	8000	4000	4000	
15	2470	103	9330	4160	5170	
9 days interval						
25	2450	103.2	9750	5000	4750	Red Corp. 10 c.c.emulsion in distilled water of dead T.B., intraperitoneally.
26	2450	103.4	12000	7250	4750	
27	—	—	—	—	—	
28	2550	103.6	7250	2750	4500	
29	2510	104.2	11000	6750	4250	
30	2460	103.4	10140	5340	4800	
31	2450	103.4	22750	12750	10000	
32	2450	103.4	14600	8400	6200	
33	2450	104	22375	14375	8000	
34	2450	103.4	14000	6500	7500	
35	2430	103.4	14330	6330	8000	
36	2470	103.4	16330	7830	8500	
37	2420	103.2	11875	7000	4875	
38	2410	103.2	12000	6330	5670	
19 days interval from last count						
58	2570	—	12750	5750	7000	1 agar culture, 48 hours old staphylococcus aureus heated to 80°C., subcutaneously.
59	2580	—	22000	15750	6250	
60	2580	—	18960	9160	9800	
61	2620	—	13200	6800	6400	
62	2600	—	12330	5670	7660	
63	2590	—	16500	8500	8000	
64	2620	—	20000	9400	10600	
65	2570	—	25750	14000	11750	
66	2580	—	10500	5250	5250	

RABBIT "F."

Day.	Weight.	Temp.	Total Leu.	Poly. Leu.	Mono. Leu.	
1	2250	102	8500	5000	3500	1 broth culture, dead staphylococcus aureus 48 hours old, intraperitoneally.
2	2230	102.2	8800	4400	4400	
3	2290	102.4	7000	4000	3000	
4	—	—	—	—	—	
5	2280	102.8	9875	2625	7250	2 c.c. broth cultures, living staphylococcus aureus 48 hours old, subcutaneously.
6	2300	103.6	11600	5200	6400	
7	2320	102.8	6250	1500	4750	
8	2320	103	10250	3250	7000	
9	2330	102.8	9750	2500	7250	
10	2350	102.4	25000	6400	18600	
11	—	—	—	—	—	
12	2350	102.2	7890	2330	5500	
13	2351	102.2	6250	1500	4750	
14	2310	100.4	4400	1400	3000	
15	2320	103	10140	2850	7290	
16	2350	102.8	12400	5200	7200	
17	2390	102.6	8160	4000	4160	
18	—	—	—	—	—	
19	2410	102.8	17000	7000	10000	
20	2420	102.8	20800	12200	8600	
21	2400	102.8	14160	7160	7000	
22	2510	102.4	13160	7160	6000	
23	2490	102.8	9800	3800	6000	
24	2460	102.8	10800	5000	5800	
25	—	—	—	—	—	

RABBIT "F"—continued.

Day.	Weight.	Temp.	Total Leu.	Poly. Leu.	Mono. Leu.	Large Monos.	Large Lymph.	Small Lymph.	Red Corp.
26 ¹	2540	103	6750	3250	3500	70	910	2520	5800000
27	2520	102.8	16125	10625	5500	110	1870	3520	6300000
28	2540	102.6	5950	2500	2750	83	687	1980	6200000
29	2500	102.7	12750	5000	7250	217	1523	5510	6100000
30	2570	102.5	10000	5000	5000	400	2100	2500	3180000
31	2590	102.4	8750	4250	4500	135	1260	3105	—
32	—	—	—	—	—	—	—	—	—
33 ²	2620	102.5	8800	5000	3800	117	1750	3963	6100000
34	2590	102.2	7250	4750	2500	125	700	1675	6100000
35	2590	102.4	12250	4250	8000	320	2009	5680	5800000
36	2570	102	11000	2750	8250	495	1815	5940	5300000
		14 days interval*							
51	2370	—	9330	4000	5330	—	—	—	—
52	2380	—	9750	4500	5250	—	—	—	—
53	—	—	—	—	—	—	—	—	—
54 ³	2420	—	9500	2960	6540	—	—	—	6100000
55	2480	—	16830	5500	11300	—	—	—	5200000
56	2470	—	13320	3160	10160	—	—	—	5900000
57	2550	—	14750	3250	11500	—	—	—	6200000
58	2540	—	1200	3500	8500	—	—	—	6000000
59	2550	—	12750	4500	8250	—	—	—	6200000
60	2620	—	14250	5000	9250	—	—	—	—
61	2620	—	10750	3500	7250	—	—	—	5600000
62	2600	—	14600	6000	8600	—	—	—	5500000
63	26.0	—	16800	7160	9640	—	—	—	5200000
64	2620	—	16000	5600	10400	—	—	—	5500000
65	2580	—	11000	3600	7400	—	—	—	5520000
66	2650	—	9000	2750	6250	—	—	—	5600000

¹ 10 c.c. sterile broth, intraperitoneally.² The cells of two rabbits' kidneys, intraperitoneally.
³ 10 c.c. T.B. extract, subcutaneously.

RABBIT "G."

Day.	Weight.	Temp.	Total Leuc.	Poly. Leuc.	Mono. Leuc.	
1	2220	101.4	3000	500	2500	The cells of two rabbits' kidneys, intraperitoneally.
2	2220	101.4	8000	4000	4000	
3	2300	101.5	6500	3000	3500	
4	2200	102	4500	2250	2250	
5	2180	102.2	4750	1750	3000	
6	—	—	—	—	—	
7	2240	101.8	4250	1750	2500	The cells of two kidneys of a guinea-pig, intraperitoneally.
8	2050	103.4	7000	3235	3750	
9	2060	102.2	7000	2140	4860	
10	2080	102.6	11000	2830	8170	
11	2120	102.6	12250	3250	9000	
12	2120	102.8	11800	3400	8400	
13	—	—	—	—	—	
14	2220	102.4	9286	3143	6143	
15	2110	102.4	8200	4600	3600	
16	2100	103	10715	4429	6286	
17	2320	102.2	11400	4600	6800	
18	2040	102.6	14000	3400	10600	
19	2220	102.6	6400	1600	4800	
20	—	—	—	—	—	
21	2260	102.6	8750	3000	5750	
22	2270	102.5	7166	3666	3500	
23	2250	102.2	8000	4500	3500	
24	2420	102.4	8500	3750	4750	
25	2310	102.6	5250	1500	3750	
26	—	—	—	—	—	
27	—	—	—	—	—	

RABBIT "G."—continued.

Day.	Weight.	Temp.	Total Leuc.	Poly. Leuc.	Mono. Leuc.	Large Monos.	Large Lymph.	Small Lymph.	Red Corp.
28 ¹	2460	102·6	7250	3750	3500	70	770	2660	5500000
29	2370	102·4	5250	2500	2750	137	605	2008	4900000
30	2470	102·4	6000	3000	3000	150	450	2400	4460000
31	2390	102·4	6750	3000	3750	112	778	2850	4800000
32	2440	102·2	10166	3880	6386	190	2283	3863	5100000
33	2470	102·2	11600	3600	8000	400	2240	5360	5100000
34	2450	102	5000	1000	4000	—	—	—	—
35 ²	2400	102·2	8000	2750	5250	263	1460	3517	5266000
36	2150	103	8500	4000	4500	135	900	3465	5200000
37	2140	103	9500	4000	5500	55	985	4510	5400000
38	2100	103·2	7380	3000	4380	87	690	3553	5100000
39	2130	105·4	7572	3000	4572	46	593	3983	5100000
40	2120	102·8	11800	4600	7200	—	—	—	5100000
		11 days interval							
52	2300	—	9660	5660	4000	—	—	—	—
53	2270	—	7250	2750	4500	—	—	—	—
54	—	—	—	—	—	—	—	—	—
55 ³	2350	—	10000	5750	4250	—	—	—	5300000
56	2250	—	5600	3400	2200	—	—	—	4900000
57	2250	—	5800	1800	4000	—	—	—	5100000
58	2270	—	7890	3660	4170	—	—	—	5200000
59	2340	—	11160	4330	6830	—	—	—	5200000
60	2380	—	18800	9200	9600	—	—	—	5700000
61	2350	—	15250	6750	8500	—	—	—	—
62	2300	—	9500	4000	5500	—	—	—	5500000

¹ 10 c.c. purified T.B. extract, intraperitoneally.² 10 c.c. extracted T.B., intraperitoneally.³ 5 c.c. extracted T.B., subcutaneously.

RABBIT "H."

Day.	Weight.	Temp.	Total Leuc.	Poly. Leuc.	Mono. Leuc.	Large Monos.	Large Lymph.	Small Lymph.	Red Corp.
1	2080	103	12500	5750	6750	—	—	—	—
2	1940	101·6	11000	5000	6000	—	—	—	—
3	2020	101·8	7000	3834	3666	—	—	—	—
4	2100	102	9380	4390	5000	—	—	—	—
5	2240	102·4	7250	3500	3750	—	—	—	—
6	2100	101·8	5500	4000	5500	—	—	—	—
7	—	—	—	—	—	—	—	—	—
8 ¹	2140	102·8	11000	3625	7375	—	—	—	—
9	1950	105	6750	2000	4750	—	—	—	—
10	1930	102·4	16572	10143	6429	—	—	—	—
11	2000	102·8	32375	11250	11125	—	—	—	—
12	2000	102·2	17660	7000	10660	—	—	—	—
13	2040	102·4	12500	4836	7664	—	—	—	—
14	—	—	—	—	—	—	—	—	—
15 ²	2170	103	12250	6000	6250	249	1687	4318	5250000
16	2050	103·4	15000	7500	7500	—	—	—	5825000
17	2100	102·2	9400	3600	5800	116	1044	4640	5400000
18	2080	102·4	12800	5400	7400	222	1850	5328	6338000
19	2080	102·4	11200	3200	8000	240	2000	5760	5800000
20	2160	102·6	19390	8000	11390	453	3400	7480	5500000
21	2190	102·6	21000	5800	15200	—	—	—	—
22	2240	103	15500	5750	9750	292	2041	7507	5700000

¹ 1 agar culture, 24 hours old living V.M. in normal salt solution, subcutaneously.² The cells of two rabbits' testicles, intraperitoneally.

RABBIT "H."—continued.

Day.	Weight.	Temp.	Total Leuc.	Poly. Leuc.	Mono. Leuc.	Large Monos.	Large Lymph.	Small Lymph.	Red Corp.
23 ⁸	2220	102·6	11660	4330	7330	225	1650	5625	5839000
24	2140	102·8	12000	4500	7500	220	1650	9130	6100000
25	2120	132·4	19330	8930	11000	262	1575	6913	5700000
26	2180	102·6	12500	3750	8750	125	937	5188	6100000
27	2240	102·6	10250	4000	6250	—	—	—	6166000
28	—	—	—	—	—	—	—	—	—
29 ⁴	2280	103	10500	4500	6000	60	840	5100	—
30	2120	103·5	7500	4500	3000	60	420	2520	5800000
31	2150	103·4	19200	7400	11800	354	2006	9440	—
32	2170	103	19750	6500	13250	265	3445	9540	—
33	2170	102·6	15800	5900	11600	424	3604	6572	—
34	2190	103·4	22250	9500	12750	637	2805	9208	—
35	—	—	24000	13500	10500	—	—	—	—
36	2270	—	17750	8750	9000	—	—	—	—
37	2270	—	18500	9000	9500	—	—	—	—

⁸ 20 c.c. sterile broth, intraperitoneally.⁴ 1 agar culture, 3 days old living V.M. in salt solution, subcutaneously.

RABBIT "J."

Day.	Weight.	Total Leu.	Poly. Leu.	Mon. Leu.	
1	2670	11330	5500	5830	0.005 c.c. diphtheria toxin, subcutaneously.
2	2660	14500	6000	8500	
3	2610	27750	20500	7250	
4	2610	29750	16500	13250	
5	2570	26000	12400	13600	
6	2500	25200	13400	11800	
7	2470	24250	12750	11500	
8	2450	38660	16330	22330	
9	2400	15600	8000	7600	
10	2420	29160	13160	16000	

RABBIT "K."

Day.	Weight.	Total Leu.	Poly. Leu.	Mon. Leu.	Red Corp.	
1	2160	13750	6500	7250	5900000	10 c.c. broth culture, staphylococcus aureus heated to 100°, subcutaneously.
2	2020	12250	8250	4000	5700000	
3	2060	17660	6000	11660	5700000	
4	2100	10500	2500	8000	5600000	
5	2180	8500	2250	6250	5600000	
6	2180	4320	1660	2660	5600000	

RABBIT "L."

Day.	Weight.	Total Leu.	Poly. Leu.	Mon. Leu.	Red Corp.	
1	2120	7000	2000	5000	—	0.02 c.c. diphtheritic toxin, subcutane- ously.
2	2250	13500	3750	9750	—	
3	—	—	—	—	—	
4	2300	11250	3250	8000	5900000	
5	2180	20000	12000	8000	6200000	
6	2160	30500	18500	12000	—	
10 a.m.						
6	—	10750	5000	5750	—	
1.15 p.m.						

The last count was taken one hour before death at 2.15 p.m. on the sixth day after injection.

RABBIT "M."

Day.	Weight.	Total Leu.	Poly. Leu.	Mon. Leu.	Red Corp.	
1	1790	9160	3160	6000	—	
2	1800	12400	5200	7200	—	
3						
4	1780	10750	4000	6750	6000000	0.02 c.c. diphtheria toxin, subcutaneously.
5	1820	21160	17660	3500	5600000	

This animal died during the next evening, *i.e.*, about eight to twelve hours after the last count.

RABBIT "N."

Day.	Weight.	Total Leu.	Poly. Leu.	Mon. Leu.	Red Corp.	
1	1860	9500	2750	6750	—	
2	1880	10500	4160	6340	—	
3						
4	1930	11800	3660	8140	5600000	8½ c.c. broth culture, 48 hours old pneumococcus heated to 80° C., subcutaneously.
5	1820	25160	10000	15160	5900000	
6	1840	17160	7000	10160	5500000	
7	1890	15750	4000	11750	5400000	
8	1920	23140	9000	14140	5800000	
9	1910	15750	5750	10000	6000000	
10	1930	14250	5000	9250	—	
11	1970	14800	4140	10660	5800000	
12	1900	23000	8000	15000	5700000	
13	1950	14000	4000	10000	5900000	
14	1950	8250	2250	6000	6000000	
15	2020	8250	3250	5000	5600000	

RABBIT "O."

Day.	Weight.	Total Leu.	Poly. Leu.	Mon. Leu.	
1	1930	6000	2000	4000	
2	1970	13250	4000	9250	
3	2000	7000	2750	4250	
4	—	—	—	—	
5	2000	12000	5250	6750	·001 c.c. diphtheria toxin, subcutaneously.
6	1950	14625	7750	6875	
7	2020	16500	10750	5750	
8	2050	16000	8750	7250	
9	2040	17160	6800	10360	
10	2070	10250	6500	3750	
11	—	—	—	—	
12	2160	8500	2500	6000	5 blood agar cultures, living micrococcus rheumaticus, intravenously in salt solution (3 c.c. fluid approximately).
13	2060	15000	10500	4500	
14	2020	11500	6000	5500	
15	2100	12400	7400	5000	
16	2030	17750	9000	8750	
17	2090	13500	5500	8000	
18	—	—	—	—	
19	2200	15000	7250	7750	1 gm. "Peptone" in 3 c.c. normal salt solution, intraperitoneally.
20	2100	8250	3500	4750	
21	2050	14000	9000	5000	
22	2080	11750	7500	4250	
23	2050	22000	12250	9750	
24	1990	9400	4000	5400	
25	—	—	—	—	
26	2190	12250	4000	7250	

RABBIT "P."

Day.	Weight.	Total Leu.	Poly. Leu.	Mon. Leu.	
1	2080	9250	2750	6500	
2	2080	9500	3000	6500	
3	2080	8250	3500	4750	
4	—	—	—	—	
5	2050	10750	6750	4000	0·25 c.c. living agar culture, 48 hours old bacillus diphtheria in salt solution, subcutaneously.
6	2000	11160	5500	5660	
7	2050	6750	2750	4000	
8	2050	13460	4800	8660	
9	2070	15750	7250	8500	
10	2090	9750	5500	4250	

RABBIT "Q."

Day.	Weight.	Total Leu.	Poly. Leu.	Mon. Leu.	
1	3230.	10750	5000	5750	
2	3110	15250	9000	6250	
3	3070	11000	5400	5600	
4	—	—	—	—	
5	2980	9000	4000	5000	1 agar culture, 48 hours old staphylococcus aureus heated to 80° C. for 10 minutes, subcutaneously.
6	2990	12750	6000	6750	
7	3040	25000	12800	12200	
8	3130	26875	8500	18375	
9	3150	17250	8000	9250	
10	3150	13360	6000	7660	

RABBIT "R."

Day.	Weight.	Total Leu.	Poly. Leu.	Mon. Leu.	
1	2190	8500	3000	5500	
2	2170	9750	5000	4750	
3	2180	12000	6000	6000	
4	—	—	—	—	
5	2180	11600	6000	5600	0.5 c.c. agar culture, 48 hours old living staphylococcus aureus in salt solution, subcutaneously.
6	2070	10570	6570	4000	
7	2090	18400	10800	7600	
8	2140	10000	4000	6000	
9	2120	11500	4000	7500	
10	2150	13800	7600	6200	

RABBIT "S."

Day.	Weight.	Total Leu.	Poly. Leu.	Mon. Leu.	
1	2280	10000	3160	6840	
2	2350	12660	4330	8330	
3	2340	15500	5750	9750	
4	—	—	—	—	
5	2460	10000	3000	7000	1 agar culture, living bacillus diphtheria 72 hours old in salt solution, subcutaneously.
6	2410	11250	4250	7000	
7	2390	17500	9500	8000	
8	2220	7750	2500	5250	
9	2410	11000	3750	7250	
10	2440	9400	3800	5600	

RABBIT "T."

Day.	Weight.	Total Leu.	Poly. Leu.	Mon. Leu.	
1	2250	8000	3250	4750	½ agar culture, 48 hours old living <i>staphylococcus aureus</i> in salt solution, subcutaneously.
2	2240	12500	4250	8250	
3	2230	10500	3250	7250	
4	—	—	—	—	
5	2250	11750	4750	7000	
6	2250	12500	8250	4250	
7	2170	15400	11000	4400	
8	2050	33750	24500	9250	
9	2050	12330	6830	5500	
10	1970	25250	14000	11250	
11	2050	17500	10160	7340	
12	1940	9750	5500	4250	

RABBIT "U."

Day.	Weight.	Total Leu.	Poly. Leu.	Mon. Leu.	
1	2040	11250	4500	6750	3 agar cultures, 48 hours old living <i>micrococcus rheumaticus</i> in salt solution, subcutaneously.
2	2020	9500	3500	6000	
3	2020	8250	4250	4000	
4	—	—	—	—	
5	2010	7500	3000	4500	
6	2100	15000	6500	8500	
7	2070	14000	7000	7000	
8	2130	13660	5500	8160	
9	2170	9800	4160	5640	
10	2210	13250	6000	7250	

RABBIT "V."

Day.	Weight.	Total Leu.	Poly. Leu.	Mon. Leu.	
1	2110	10000	5250	4750	3 agar cultures, 48 hours old living <i>micrococcus rheumaticus</i> in salt solution, subcutaneously.
2	2120	6000	2000	4000	
3	2130	8500	3750	4750	
4	—	—	—	—	
5	2150	9000	3000	6000	
6	2200	13660	6830	6830	
7	2270	14600	7200	7400	
8	2290	12250	5750	6500	
9	2190	15500	6000	9500	
10	2140	10000	4750	5250	

A CASE OF SUBDIAPHRAGMATIC AND HEPATIC ABSCESS CONSECUTIVE TO MEDITERRANEAN FEVER.

BY J. W. H. EYRE, M.D., AND J. FAWCETT, M.D.

IN writing of suppuration, the statement has been made by one of us¹ that almost all the micro-organisms pathogenic for man possess the power, under certain conditions, of initiating purely pyogenic processes in place of, or in addition to, their so-called "specific actions"—the pneumococcus, and the tubercle bacillus, being quoted as familiar instances in support of the contention. Additional support is afforded from time to time by evidence obtained from the clinical laboratories of our hospitals during the routine examination of pus from various lesions. Thus at the present time *B. influenzae* is one of the few pathogenic microbes that has not been conclusively shown to be capable of taking on a pyogenic rôle. Until recently *Micrococcus melitensis* was regarded as another exception, for although in many cases of Malta Fever serous effusions have been noted (and recorded as probably directly due to the activity of this coccus), suppurative lesions, possibly on account of their extreme rarity in this association, had not so far been attributed to the coccus. During the summer of 1903, some observations on the results of the intracranial and subdural injection of *M. melitensis* into rabbits and guinea-pigs indicated

¹ Art. Pyogenic Bacteria (Eyre). Quain's Dictionary of Medicine. Third Edition. 1902.

the probability, however, that localised collections of pus might well result, in man, from the active multiplication of this organism. The lesions produced at, or near, the seat of inoculation in the lower animals varied in subacute and chronic infections from localised membranous exudation to suppurative meningitis and small pockets of pus.

Later on, in a case which recently came under our notice, the evidence of the causal relationship of *M. melitensis* to the suppurative process was so clearly established as to encourage us to record it in the hope of eliciting further observations on the subject from those who have better opportunities than ourselves of investigating the disease as it occurs in man.

To Mr. Jacobson, under whose care the patient was admitted, we tender our thanks for his kind permission to publish the details of the case.

CLINICAL OBSERVATIONS.

G.T., æt. 38, a coal porter, was admitted into Naaman ward, Guy's Hospital, on July 25th, 1903, suffering from abdominal pain.

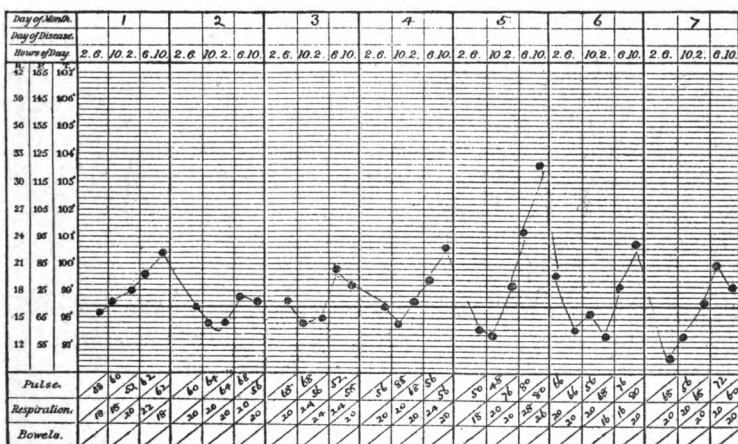
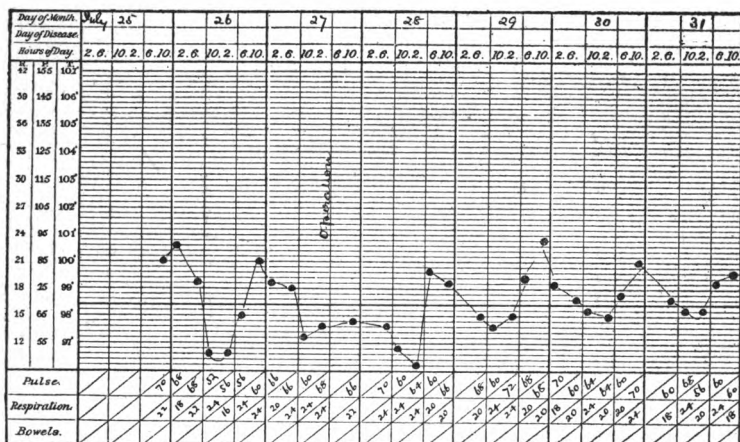
The patient had been in India for six years and in Malta for two years, and in the latter place he contracted Malta Fever.² Since this time he had no illness until the one for which he was admitted.

History of present illness.—Eleven days before admission, that is on July 14th, he suddenly felt pains in the stomach while at work. He continued at work the following day, but was unable to do so afterwards. The pain was continuous and worse on standing, or moving about.

Condition on admission.—A smooth round tender mass, thought to be connected with the left lobe of the liver, was felt in the epigastrium. The liver dulness extended upwards, higher than normal, but the lower edge of the right lobe was not palpable. On inspection, the right hypochondrium appeared to be somewhat more full than the left. A leucocyte count gave 11,400 per c.mm.

The temperature varied from subnormal to 100·6° F.

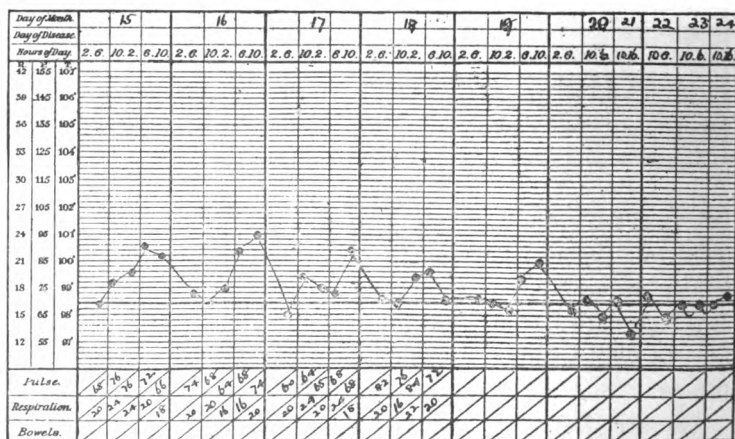
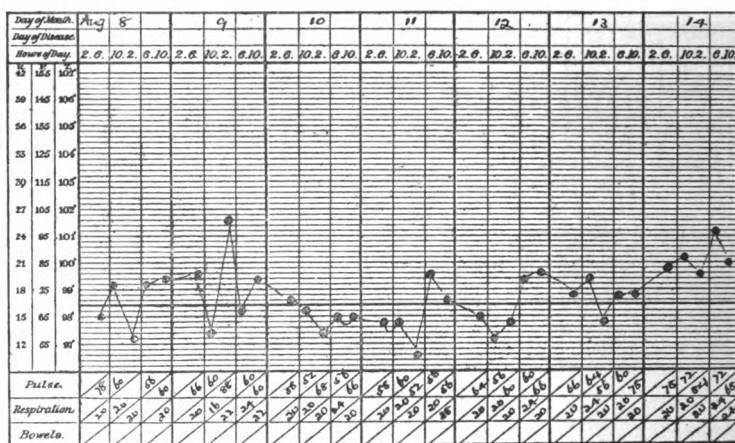
² Unfortunately precise data on these points are lacking.

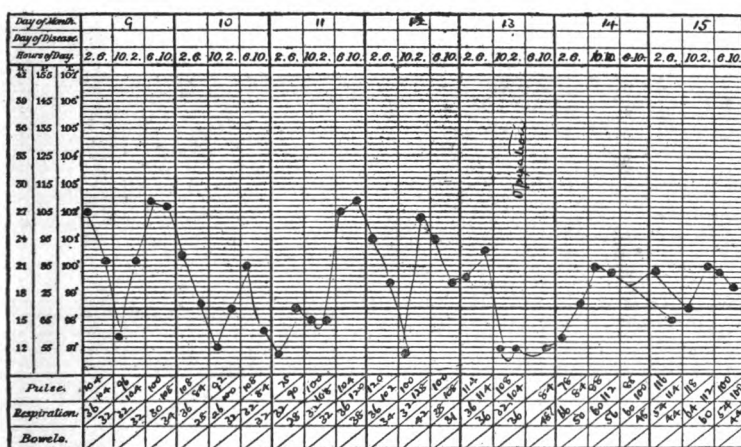
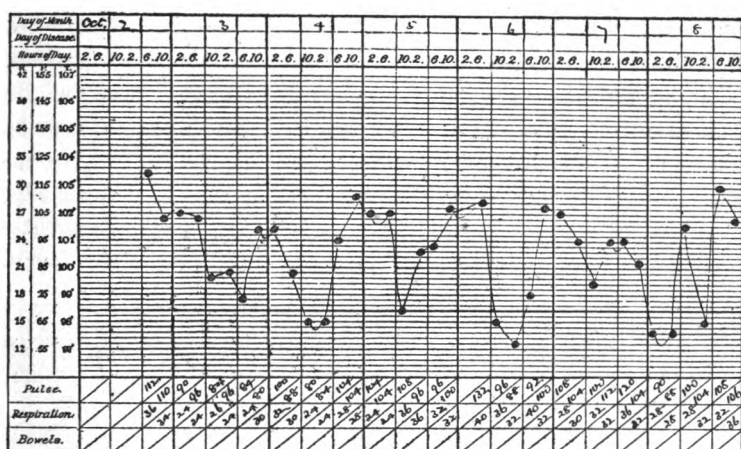


July 27th. Mr. Jacobson explored the swelling in the epigastrium and opened an abscess which was not in the liver, but which formed a localised intraperitoneal collection. From this date the patient's progress was on the whole satisfactory, and he was discharged on August 25th, 1903, with the wound almost healed.

October 2nd. The patient was re-admitted owing to the abdominal pains having returned within the last fourteen

days. He had also had a "shivering fit," followed by profuse perspiration, every day during this period. He looked sallow and ill, much more so than on his former admission, and his temperature chart (*quod vide*) showed larger morning and evening variations than previously. There was marked fulness in the right hypochondriac and epigastric regions, and the upper edge of the liver dulness presented a curved line running convexly upwards from the level of the seventh rib behind, to the fourth



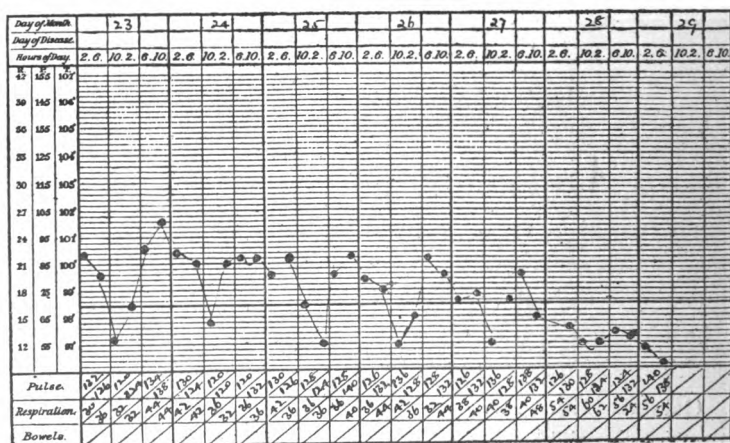
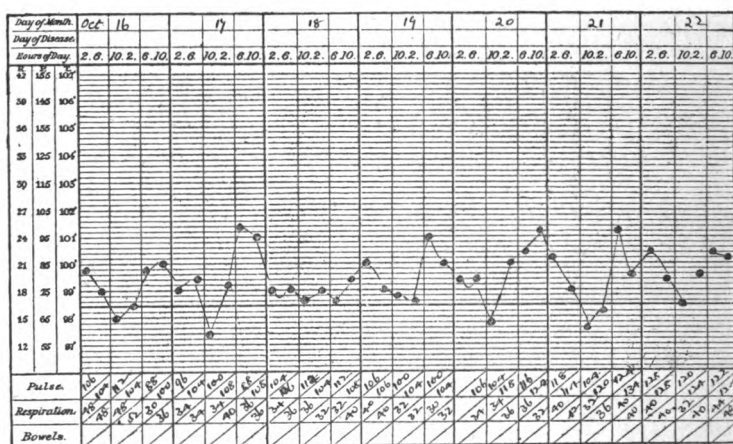


rib in the middle line in front. By the request of Mr. Jacobson one of us again saw the patient and gave the opinion that the patient was suffering from a subdiaphragmatic abscess.

October 8th. A specimen of blood was collected from the lobe of one ear and the serum obtained therefrom yielded a positive reaction when tested against the *M. melitensis*.

October 13th. Mr. Jacobson again operated, on this occasion, through the right pleura, and after resecting a portion of a rib

and taking careful precautions to shut off the pleural cavity, he incised the diaphragm and opened a large abscess cavity beneath it. The pus contained therein was thick and of a greenish colour. No indication was obtained at the operation as to the cause of the abscess. Although the temperature was somewhat lower after the operation, it still showed variations from subnormal to 101° F. The patient's condition did not materially alter, and he died sixteen days later, on the 29th October, 1903.



Dr. A. E. Boycott (Gordon Lecturer on Experimental Pathology) performed the autopsy, thirteen hours after death, and the following abstract is from the notes made by him :—

Inspection, 1903, No. 409.—*Lungs and pleura*.—Right side.—Recent pleurisy with thick lymph over the lower lobe and between the lobes. In the lower lobe was a large irregular cavity with ragged walls, full of thick pus and broken-down lung tissue; the contents were odourless. A similar cavity was present in the lower part of the upper lobe. Left side.—In the lower part of the upper lobe was a small abscess cavity the size of a pea, containing thick, yellow pus, and higher up another small abscess.

Abdominal viscera.—The diaphragm on the right side was firmly fixed by recent adhesions to the corresponding lung, and to the liver, and in the middle of the adherent area was a hard white scar. There was no evidence of any recent extension of inflammation from the liver to the right lung.

The anterior surface of the right lobe of the liver was firmly adherent to the scar of the first operation wound, and below this a large cavity extended into the substance of this viscus.

The cavity had cartilaginous walls and thick, yellow, slimy contents. In the right lobe there was also a number of abscesses, irregular in outline and without any definite walls, and filled with thick yellow pus. Microscopically, sections of the liver showed large areas of necrotic liver substance, the cavities having no definite limiting membrane.

BACTERIOLOGICAL OBSERVATIONS.

A specimen of the pus was removed at the first operation (July 27th) for bacteriological examination. No suspicion was apparently entertained at this time of a connection between the previous attack of Malta Fever and the existing abscess, and the pus sent to the laboratory was merely stated to have been removed from an "abdominal abscess." The specimen was consequently examined in the routine manner, that is to say, a coverslip film preparation stained with carbolic methylene blue, and another stained by Gram's method, were examined microscopically. As

no micro-organisms could be detected a third film was stained by the Ziehl Neelsen method and examined for the presence of tubercle bacilli, but again the result was negative. A tube of nutrient broth was inoculated with a small loopful of the pus and another tube containing an ordinary nutrient agar slant was inoculated with a similar amount of pus. The tubes were incubated at 37° C. and examined at intervals of twenty-four hours until the end of the fourth day, when, being still sterile, they were destroyed, and the negative result recorded. If ordinary pyogenic cocci or *B. coli* had been present this procedure would almost certainly have revealed their presence.

About two months later, on October 8th, the clinical symptoms prompted the collection of a specimen of the patient's blood in order that the serum might be tested for the presence of agglutinins. The serum diluted with sterile saline (0·5 per cent.) solution was tested against an eighteen hour broth cultivation of *B. typhosus* and a forty-eight hour broth cultivation of *M. melitensis*, the latter being previously filtered through sterile filter paper to remove any small preformed masses of cocci. The serum dilutions employed in each case were 1:20 and 1:200 and the time of observation limited to half an hour. The test as carried out against the typhoid bacillus was completely negative, even the 1:20 dilution showing no evidence of clumping of the bacilli at the end of thirty minutes.

When, however, the *M. melitensis* preparations were examined the cocci were found to be completely agglutinated into large masses—no cocci could be seen free in the field—in both the 1:20 and 1:200 dilutions within twenty minutes, while the control preparations of cocci and sterile broth examined at the same time showed active Brownian movement without any trace of “clump” formation.

The specimen of pus removed for examination at the second operation (October 13th) was received in the bacteriological laboratory as “specimen of pus from liver abscess.” Consequently fresh films were examined microscopically for the presence of amœba dysenteriae, but with negative results. Microscopical examination of stained film preparations showed the presence

of a very few minute spherical bodies which did not retain the stain when treated by Gram's method, but no other micro-organisms could be detected.

A glycerine agar slant cultivation was then made, and in view of the small number of organisms present a large platinum loop was employed and about two milligrammes of the pus planted on to the surface of the medium; a broth culture was also made and both these cultivations were incubated aerobically at 37° C. (Two control cultivations were prepared and incubated anaerobically at 37° C., but as no obligate anaerobic organisms made their appearance, the culture tubes were discarded after eight days' observation.)

At the end of some forty-eight hours' incubation two small colonies had developed upon the agar slant culture, which on subsequent microscopical examination proved to be composed of minute micrococci similar in size to those noted in the film preparations made direct from the pus. When sub-cultivated upon various media and tested against the highly diluted serum of a rabbit immunised to an authenticated Malta Fever coccus the organism proved to be indetical with *M. melitensis*, and is still kept under cultivation in our laboratory collection as strain No. 5.

No other micro-organisms were detected, although the original cultivations were kept under observation for a considerable period.

At the autopsy, on October 30th, the specimens secured for bacteriological examination included (1) blood from the right ventricle of the heart, (2) swabbings of sero-pus collected from the sinus leading from the operation wound to the upper surface of the liver, (3) pus from the liver abscess, and (4) the entire spleen.

The results obtained may be briefly summarised as follows:—

1. The heart blood on cultivation gave evidence of the presence of bacillus coli; but *M. melitensis* could not be detected.
2. Cultivations were established upon glycerine agar and in broth. After incubation at 37° C. for forty-eight

hours a mixed growth resulted in each of the tubes, consisting of *B. coli*, *Staphylococcus aureus*, and *S. albus*, and *M. melitensis*.

3. Cultivations prepared from the liver pus yielded a mixed growth of *B. coli*, *Staphylococcus albus* and *aureus*, and *M. melitensis*.
4. The spleen surface was thoroughly seared with a red hot iron and incised with a sterile knife. Several glycerine agar slants were then inoculated each with a loopful of the spleen pulp taken from the centre of the organ. Each tube after incubation yielded a pure growth consisting of a few colonies, usually two or three (none more than three), of *M. melitensis*.

In considering these results, it may be concluded that the presence of *B. coli* was in all probability the result of the usual post-mortem infection from the alimentary canal, for thirteen hours had elapsed between death and the post-mortem inspection, and during that interval the body was not kept in an ice chamber.

Further, in view of the result of the bacteriological examination of the pus obtained at the second operation (October 13th), the demonstration of *Staphylococcus aureus* and *S. albus* in pus obtained at the autopsy, sixteen days later, may fairly be assumed to indicate nothing more than secondary infection of the sinus and liver cavity from the skin surface around the wound.

Therefore, by a process of exclusion, supported by analogy derived from animal experiment, *M. melitensis* remains as the organism standing in causal relationship to the pyogenetic process observed in this case.

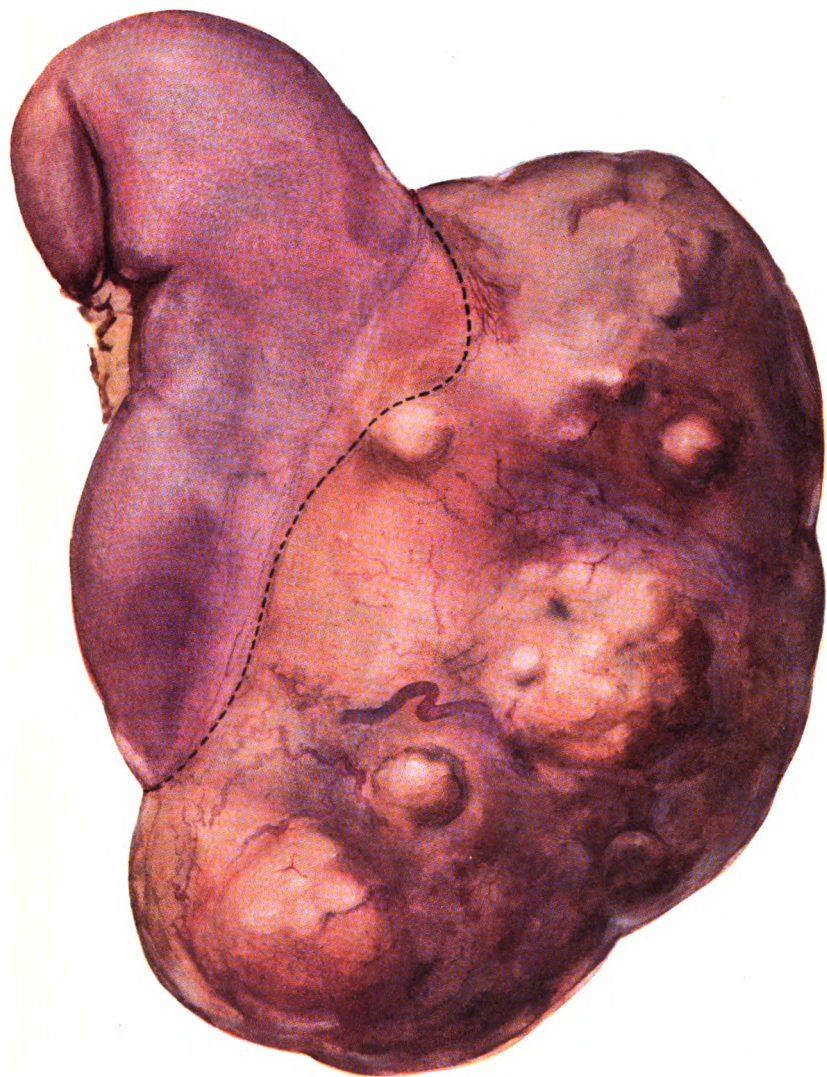
[The expense of the inoculation experiments necessitated by this investigation was defrayed out of a grant from the Royal Society.]



FIGURE 11

FIG. 5.

Growths of the Kidney and Adrenals.



Four-fifths natural size.

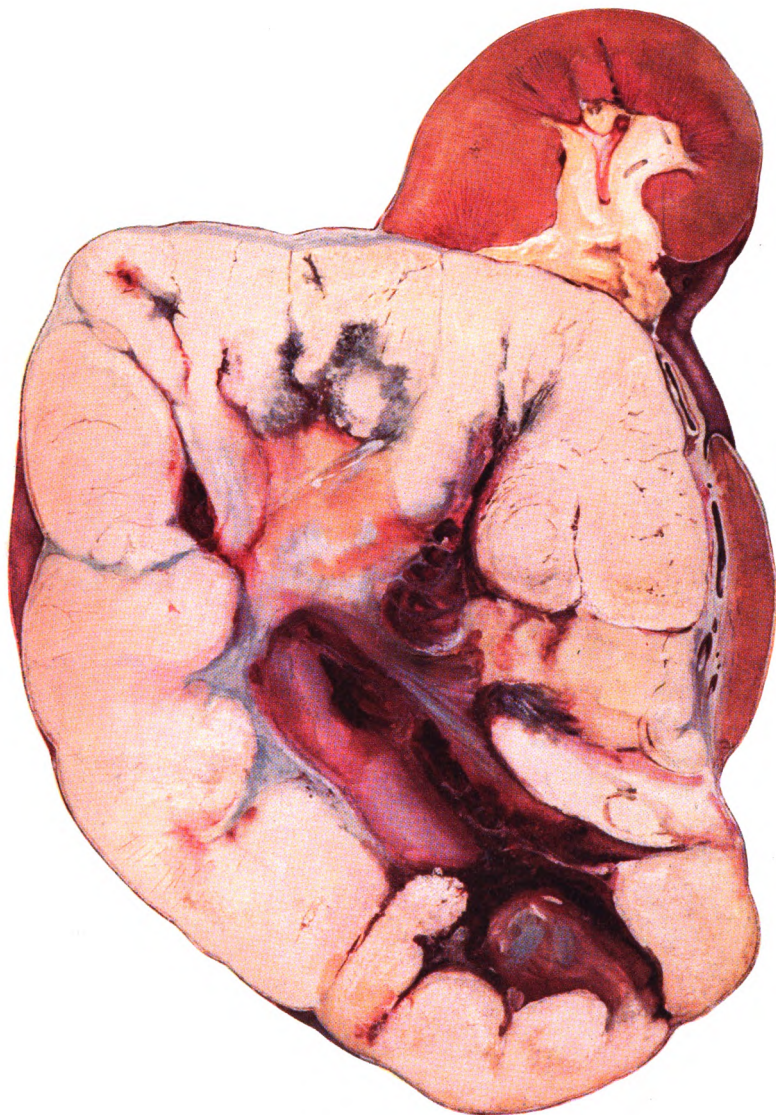
FIG. 5.



PLATE 10

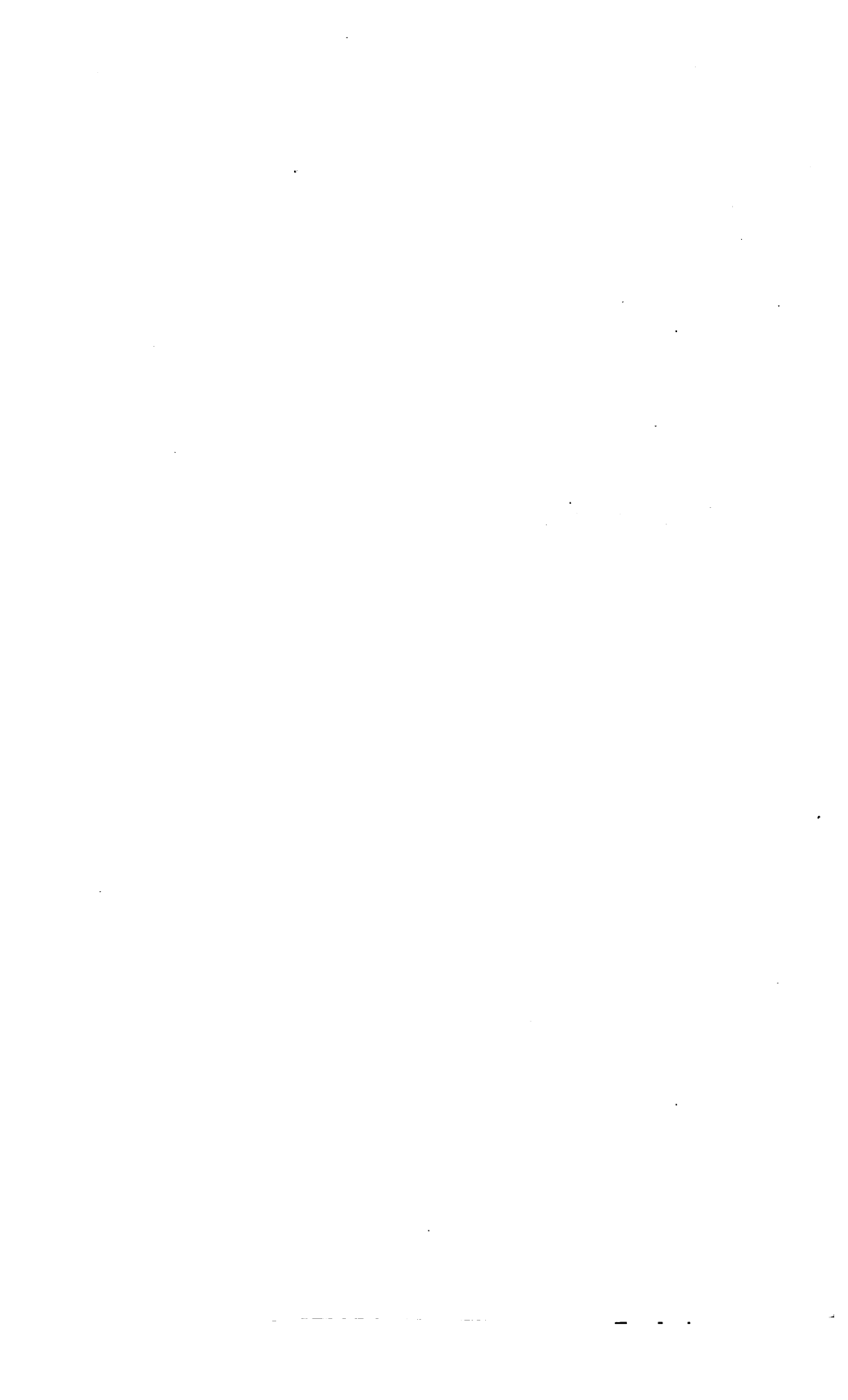
FIG. 10

Growths of the Kidney and Adrenals.



Four-fifths natural size.

FIG. 6.



DESCRIPTION OF FIGS. 5 AND 6.

These figures represent the kidney removed by Mr. Golding-Bird from case 70. The upper end and inner border of the kidney are intact and healthy; the growth is situated in the lower end. It possesses a definite capsule which is continuous with the kidney capsule, with a fibrous boundary which marks it off sharply from the renal tissue, and with the septa which divide the growth itself into nodules. Thus the growth consists of a collection of rounded masses lying beneath the kidney capsule, and separated from one another and from the renal tissue by fibrous partitions. Externally (Fig. 5) some of these nodules show a tendency to herniate through the capsule, but none have as yet penetrated it.

The pelvis is not invaded, but is compressed, and those calyces which pass to the lower end of the kidney can be seen in Fig. 6 as flattened tubes running outside the capsule of the growth. The centre of the growth is occupied (Fig. 6) by a very irregular cavity with fibrous walls, which contained old blood-clot. It was not possible to demonstrate any connection between this cavity and the pelvis. A probe can be passed in several places nearly to the surface of the growth in the neighbourhood of the bosses shown in Fig. 5. It is possible that this is a hæmatoma of some standing. In other parts the growth, which was naturally whitish, was stained presumably by old blood pigment.

At the upper left hand edge of Fig. 6 the growth rises abruptly from the kidney, but over the junction represented by a dotted line in Fig. 5, the transition is gradual, and on making a small incision it can be demonstrated that here the kidney forms a kind of cap to the tumour, and that the renal tissue gradually thins out over it, till finally only capsule is left.

The large veins which are seen in an empty condition in the capsule have been found in other cases a source of great difficulty to the operator.

The growth, as a whole, is a typical instance of the nodular form as contrasted with the infiltrating (p. 237), and shows the macroscopic characters which are connected with adrenal rest growths (p. 241).

Sections were examined from two different parts of the growth, but although the appearances suggested an adrenal origin, they were not definite enough to justify a decided opinion, and the examination must be considered inconclusive. The consideration of the macroscopic, microscopic, and clinical evidence together is in favour of this origin, but does not prove it.

GROWTHS OF THE KIDNEY AND ADRENALS.

BY OWEN RICHARDS, M.B., B.CH.

In the last twenty years a large number of cases of these growths have been recorded, and the technique of nephrectomy has been much improved. Nevertheless, the results of treatment compare unfavourably with those obtained in other forms of malignant disease. This is due partly to the difficulty of making a sufficiently early diagnosis, partly to the absence of any certain indications as to which cases are fit for operation. For it is clear that while some forms of growth persist for years without metastases, and can be totally and permanently removed others disseminate widely in the course of as many months, and are inoperable before they become evident. What is needed at the present time is not so much the description of rare and anomalous forms of growth (interesting as these are from the point of view of pure pathology), as the determination of the life-history of those types on which pathologists are more or less agreed. If these could be brought into relation with clinical signs, and the means of diagnosis so extended and applied as to ensure their recognition in an early stage, vastly better results could be obtained. As Dr. Kelynack remarks, "The indiscriminate removal of renal growths is neither reasonable nor reassuring"—but at present the means of discrimination are inadequate.

In the present paper those cases of primary renal and adrenal growths have been collected which occur in the records of Guy's Hospital between the years 1826—1904. Perirenal, secondary, and small innocent growths have been excluded. The clinical and post-mortem records are usually full; histological examination is lacking in the earlier cases, and not always decisive, for reasons given below, in the later ones. The value of the series is therefore chiefly clinical, and as the majority of cases were not operated on, it gives a fair

picture of the natural course of the disease. The cases have the great advantage of being entirely unselected, but their number (seventy), is a small one to draw numerical conclusions from.

As regards the arrangement an attempt has been made,

A. To classify the ordinary forms of growth, and give a short account of what is known of their life-history and peculiarities.

B. To consider the clinical course of the cases which have occurred in Guy's Hospital, with special reference to the duration of symptoms, and the time and manner in which metastases occurred.

C. To review the available means of diagnosis, and the results which have been obtained in the past; and to formulate some practical conclusions.

References to the authorities quoted, and to others, will be found in an appendix, arranged alphabetically. A short note of the contents of each paper is given (instead of its actual title), and the year in which the communication was published; the references to pages which follow authors' names refer to their original papers, and where several papers are by the same author a number is prefixed to shew which is referred to. A good bibliography up to 1898 is to be found in Dr. Kelynack's book. This list of references makes no claim to completeness, but contains some of the more recent work accessible in London.

The most important contributions that have appeared lately on this subject are those of Walker,¹ Heresco,² Kelynack,³ and Morris.⁴ The two last named authors treat the subject as a whole; Heresco deals with the question of operation and its results, as shewn by a series of 165 nephrectomies between 1890 and 1899; and Walker reviews the pathology, diagnosis and treatment of the growths occurring in children, as illustrated by 142 cases and 74 nephrectomies. A vast deal of pathological work has also been done in Germany, especially on those tumours which are referred to the growth of adrenal rests.

1. "Sarcoma of the Kidney in Children." *Annals of Surgery*, xxvi. 529, 1897.
2. "De l'intervention chirurgicale dans les tumeurs malignes du rein." *Thèse de Paris*, 1899.
3. "Renal Growths." 1898. Young J. Pentland, Edinburgh and London.
4. "Surgical Diseases of the Kidney and Ureter." 1901. Cassell & Co., London.

A.—PATHOLOGY.

CLASSIFICATION.

The classifications suggested by Mr. Paul and Mr. Reginald Harrison are founded on a division into congenital tumours, and those of adult origin. Since many tumours which originate in embryonic misplacements make their first appearance in adult life, this criterion is a difficult one to apply with certainty. Dr. Kelynack adopts Dr. Newman's grouping, which is as follows :

- (A) *Benign.* 1. Fibromata :
- a* Minute growths, "Nephritis interstitialis tuberosa."
 - b* Simple fibroma.
 - c* Fibro-cystoma.
 - d* Fibro-myoma.
 - e* Fibro-lipoma.
2. Osteoma.
3. Lipoma.
4. Hæmatangioma.
5. Adenoma : papillary and alveolar.
6. Papilloma.
- (B) *Malignant.* 7. Carcinoma :
- a* Encephaloid.
 - b* Schirrus.
 - c* Colloid.
 - d* Epithelioma.
 - e* Cylindroma.
8. Lymphadenoma.
9. Sarcoma :
- a* Round-celled.
 - b* Spindle-celled.
 - c* Alveolar sarcoma.
 - d* Angiosarcoma.
 - e* Adenosarcoma.
 - f* Myosarcoma.
 - g* Myxosarcoma.

He suggests for use in the future some classification on the principle of that recommended by the Royal College of Physicians

(1896), in which tumours (other than cysts) are divided into sarcoma, carcinoma, those composed of some form of fully developed connective tissue, and those resembling a complex tissue.

In the present paper a classification on the principle of Hanse-mann's has been employed, based on the origin of the growths, and the extent to which they differ from the fully developed tissue.

ADRENAL GROWTHS.

Adenoma and "adrenal rest tumours" of the adrenal.

Sarcoma (? carcinoma).

RENAL GROWTHS.

I. From the connective tissue framework.

a Growths of fully formed connective tissue.

b Sarcoma.

II. From the vessels.

a Angeioma.

b Endothelioma.

III. From the epithelium.

a Adenoma—papilloma.

b Carcinoma—epithelioma.

IV. From included tissues.

"Embryonic" mixed tumours of children.

Adrenal rest tumours.

This method of division is simple and has the advantage of clearly defining the growths in Class IV., which are responsible for a good deal of confusion in the past, as it is difficult to classify them on any purely histological basis. It could, perhaps, be further simplified by disregarding the angeiomata, and grouping the endotheliomata under the heading of sarcoma.

DIFFICULTIES PECULIAR TO RENAL GROWTHS.

One of the causes of the confusion which reigns over the pathology of these growths is the situation of the kidney at a kind of developmental cross roads, so that different tissues become mixed up and included in one another.

The origin of the adrenals is not absolutely certain, but according to Keith, the medulla is developed from the cell basis of the semi-lunar ganglion, and the cortex from the endothelium covering the

Wolffian ridge, which subsequently invades and replaces the medulla. The pelvis and collecting tubules of the kidney arise from a diverticulum of the Wolffian duct near its entrance into the cloaca; this grows upwards to join the rest of the organ, which is formed from the intermediate cell mass.

In the foetus of two or three months, according to Grawitz, the adrenal is bigger than the kidney, and lies round it, separated only by the cells which give rise to the capsule, and covering the whole of the organ except the hilum and part of the surface below this. Later, the two organs are about the same size, and ultimately the adrenal becomes a much smaller mass applied to the head of the kidney. The kidney is originally lobulated, and the clefts between the lobules gradually close. As the adrenal shrinks upwards portions of its substance become fixed in these clefts, or to the surface between them, and form "rests" of adrenal tissue in the kidney capsule; while if the fixation is at the bottom of a cleft, they may be actually embedded in the renal substance. Grawitz gives instances (i. p. 826-7) where these rests were found in clefts that were still recognisable. Sometimes small masses of perirenal fat are included in the same way, either alone or in company with adrenal rests (Horn, p. 209).

In the same way renal or Wolffian tissue is (less commonly) carried up into the substance of the adrenal, as described by Ricker, and well illustrated by case 53 of the Guy's Hospital series; while in the reverse direction Wolffian rests are usually, adrenal rests fairly commonly, and renal rests quite conceivably carried down by the descent of the genital gland to any point between the kidney and the testis or ovary.

A second source of misplaced tissue in the kidney is found in the tubules of the suppressed Wolffian body, which are considered by many authorities to be the origin of "embryonic" tumours arising in childhood. In this way there are at least two possible extra-renal origins for a tumour which is clinically renal, viz., one adrenal and the other Wolffian.

The other main difficulty is the fact that microscopical examination often affords no clue, or a misleading one, to the nature and clinical history of a renal growth. Allen and Cherry, after a detailed

examination of 29 specimens, conclude that "a histological structure closely resembling carcinoma, with alveolar arrangement, large epithelial cells, and bold nuclei, is compatible with clinical innocence. The macroscopic and clinical characters of a growth are of especial importance in estimating the malignity of any renal tumour."

Mr. Morris is equally decided on this point (iii., p. 1336). "Pathologists and practical microscopists have in many instances given totally different interpretations of the same tumour. What one regards as a typical renal tumour of accessory adrenal origin, another speaks of as a spheroidal-celled carcinoma, another as an endothelioma, another as an epithelioma with clear cells, another as a renal adenoma; and again other observers have described them as primary angiosarcoma, or alveolar sarcoma, or lymphadenoma." There is plenty of testimony to this confusion, which chiefly concerns two classes of tumours, the embryonic and the adrenal rest growths. The synonyms of the latter are given above, the former are variously described as rhabdomyosarcoma, sarcoma, carcinoma, myxoma, adenoma, and various combinations of these. It follows that any generalizations based on the clinical course of, *e.g.*, all the cases described in the literature as carcinoma would be quite valueless, since they would include instances of embryonic sarcoma, adrenal rest tumours, and true carcinoma under a common heading. The only cases available for drawing conclusions from are those in which a pathologist familiar with these peculiar difficulties has examined the case as a whole, clinically, in the post-mortem room, and microscopically, and has satisfied himself that it belongs to a certain class. Even then the personal equation plays a large part.

ADRENAL GROWTHS.

1.—ADENOMATA AND ADRENAL REST TUMOURS OF THE ADRENAL.

Adenoma occurs (Rolleston i.) most commonly as multiple small yellow nodules in the cortex, occasionally as a larger single mass. Its structure is usually the same as that of the cortex, but it is commonly in a more advanced condition of fatty infiltration. Occasionally it breaks down centrally to form a cyst (Craufurd, Rolleston iii., Bosanquet) or a hæmatoma occurs in it. Dr. Rolleston (ii.)

records a fibro-adenomatous form with large vessels, fibrous stroma, and no fatty infiltration. In the Guy's records they are noted 27 times in the years 1891-8 (*i.e.*, 1 in 146 cases), but their frequency varies with the care with which they are looked for. It has been suggested that they cause an adrenal intoxication, rise of blood pressure, and cerebral hæmorrhage; but in these 27 cases cerebral hæmorrhage only occurs twice, and the absence of symptoms is to be expected from their position in the cortex (of which the extract is inactive), and their advanced fatty infiltration. True cysts and tubes with epithelial lining may occur in them, and in the growths which arise from them, and have been variously explained as reversions to a tubular type, the results of degeneration or hæmorrhage, or renal inclusions. The last have already been referred to (p. 221). The growths which arise from adenomata are of the kind described below as "adrenal rest tumours," and since they occur more frequently in connection with adrenal rests in the kidney, and there present precisely the same characters, they will be described in detail under that heading. Unless they invade the kidney, however, they do not give rise to hæmaturia, but present the symptoms described under sarcoma of the adrenal (p. 224).

Grawitz (*ii.*, p. 830), gives a good instance of a large lobulated growth of the adrenal, without involvement of the corresponding kidney, but with metastases in the lungs and liver. This presented a typical adrenal structure. Kelly describes one (p. 290) which had involved the surrounding tissues and infected the blood stream. Mr. Mayo Robson successfully removed one the size of an orange. In this case pain had been present for a year, and tumour for a month, but the symptoms were not definite enough to allow of the diagnosis being made. The patient was well nearly two years later. A wedge from the kidney was removed as well as the tumour. Blackburn reports a tumour weighing 6 lbs., with no metastases, which was probably of this character.

The difference between adenomata, and "adrenal rest tumours" of the adrenal which have not yet become malignant, is purely one of size. Instances are given by Beadles (*iv.*), Berdez, Blackburn, Kelynack (*i.*, *ii.*), Moxon, Bland Sutton (*ii.*), and Wigglesworth, of masses of this kind in the adrenal. As regards their course the

cases show that they may reach a considerable size without giving rise to metastases, but that they may ultimately become malignant, and kill the patient by dissemination.

II. SARCOMA AND CARCINOMA.

The usual kind of growth in the adrenal is a round or mixed celled sarcoma, but there is almost as much uncertainty about these growths as about those of the kidney. Otto Ramsay, who has collected 67 cases, finds the published diagnoses unreliable. He notes that the proportion of "carcinoma" is much greater in the earlier records, and probably many of the cases so described would now be called adrenal rest tumours. The difficulty is illustrated by Greenhow's case, where the Committee of the Pathological Society reported that it resembled a medullary carcinoma, but was actually a sarcoma. However, Manasse is clear that his case (28), is a carcinoma, and Ritchie and Bruce describe one with an invasion of the lymph glands and pleural lymph channels. The question is an open one, but at any rate there are not data enough to discuss the forms separately. Considered together, it appears that they occur at any age, and that the cardinal symptoms are :—

1. Languor, weakness, wasting.
2. Gastro-intestinal disorders, nausea, vomiting, diarrhœa, or constipation.
3. Pain referred in various directions. This does not radiate to the groin and testis, but rather straight round the abdomen, and by involvement of the phrenic to the shoulder tip. (Mayo Robson).
4. Tumour, usually not retrocolic.
5. Skin changes. In 3 of the 37 that Ramsay found a clinical account of, there was bronzing, and in 9 some change of tint, *e.g.*, to a dirty yellow.

Ramsay found that there was no subnormal temperature, as Berdach suggests. Hæmaturia was accidentally present, and complicated the diagnosis in two cases. In 5 the symptoms were wholly respiratory, in 8 there were none referable to the adrenals. He puts the duration at 6-10 months, Affleck and Leith in a smaller series, at 2-5 months. Clearly then these growths usually run a rapid course

and are hard to diagnose, in none of the operated cases (Morris) was a diagnosis made beforehand. Their tendency to extend upwards rather than downwards causes respiratory symptoms to occur frequently (Sloukoventoff, Turner, West). Involvement of the kidney by a growth primary in the adrenal seems to be relatively rare, the kidney capsule offers a remarkable resistance to the passage of growth either inwards or outwards. As regards metastases the histories are rather incomplete, but they seem to occur frequently and widely.

Apart from discoloration several curious skin changes accompany certain adrenal growths.

In the Museum of the Royal College of Surgeons is a specimen (3518 E), found in a woman of 32, who suffered from mania and epileptic fits; and it is noted that her face and extremities were so thickly covered with hair that a razor had been used.

In Knowsley Thornton's well-known case (iii.) the patient, a woman aged 36, was covered all over with black silky hair and had to shave her face. An ovariectomy had been performed six years before. Knowsley Thornton removed the tumour, which weighed some 20 lbs., and after various complications, caused by an abscess which burst into the lung, the patient made a complete recovery, and wrote nine months later to say that she was now normal in appearance, had gained two stone in weight, and could walk or drive any distance. Unfortunately she died of intraperitoneal recurrence two years after the operation.* The structure of the tumour "reminded the observer of the structure of an adrenal," and in the preceding case the description of large granular epithelial cells in alveoli separated by capillaries suggests that both tumours probably belong to the class of adrenal rest growths; though these effects are more likely to be due to their chronicity than their nature. An instance of the successful removal of such a growth is given above (p. 223), and the fact that in Knowsley Thornton's case the symptoms had existed so long, and the growth reached so great a size, and yet operation did so much good, points to such cases as being among those suitable for interference.

Another small group of cases is recorded by Pitman, Colcott Fox, and Dickinson. The patients, in each case girls of 2 or 3, had

*MS. note in R.C.S. Museum Catalogue, 3507 B.

become in the last year or so dull in intellect, fat, and covered with hair. They died with signs of exhaustion and vomiting.

Dr. Walker, of Budleigh Salterton, has kindly sent me notes of the following clinically similar case, in which unfortunately the precise origin of the growth was not determined :—

The patient, a girl, was healthy up to the age of 5, when she became very stout and a growth of hair appeared on face, chin, and pubes. At 7 years this had grown to the average length of that of a man of 20, and the pubic hair was that of an adult. She then lost flesh on the face and limbs, her abdomen swelled, and a tumour was observed in the left hypochondrium. Slight albuminuria, no hæmaturia. Eighteen months later she was admitted to hospital. She then had the facial aspect of a young man of 20, with black silky beard and whiskers. Abdomen distended, urine as before. Ascites was present, and she was tapped, whereupon the tumour and an enlarged liver became palpable. She died a few days later. Post-mortem: Thorax not examined. Liver enlarged and full of growth. Left kidney, the size of a cocoanut, consisting entirely of new growth. Right kidney about half this size and nearly destroyed by growth. Several mesenteric glands enlarged. Pelvic organs healthy, and normally developed for the age.

No proper examination of the growth was made, but the symptoms referred to above suggest strongly that the growth was primary in the adrenal, since obesity and growth of hair are not found with the common renal growths of children, and the age, sex, and course correspond closely with the recorded cases referred to above.

Amongst the Guy's cases Case 44 is of great interest (p. 317).

The nature of this is obscure; the fact that the masses in the skin decreased in size, and that one, 5 inches across, nearly disappeared, is against their being secondary deposits of an ordinary kind. The pathology of mycosis fungoides is hardly worked out. Bowen gives the report of a post mortem examination of one typical case of this disease in which the only internal lesions discovered were a growth extending along the spermatic cord from both testes to the pelvic brim, and nodules in one kidney and the ascending mesocolon. In this case the enlargement of the testis was the first symptom, and in another, not examined post-mortem, it was an early one. All the growths in the first case had the same character as those in the skin—and their occurrence along what may be called the "adrenal rest tract" is suggestive—but nothing more. In this case also some of the cutaneous growths had sloughed off leaving pigmented scars.

A rather similar case is one which Clauffard records :—

The patient, a man of 37, was admitted for chronic diarrhoea of a year's duration. He had loss of appetite, vomiting, and lumbar pain. His urine was normal, and no tumour could be felt. In his skin were 110-120 nodular tumours, in size from a lentil to a nut, sessile, broadly pedunculated, mobile, and asymmetrical. The first appeared 5 years ago. In the skin, which had an earthy colour, were some 20 irregular brownish pigment patches the size of a sixpence, which had increased in number during the last 3 years. No bronzing. The patient wasted and died in 5 months. Post-mortem: The nodules, microscopically fibromata, were not connected with the nerves, *i.e.*, were not neurofibromata. Both adrenals were enlarged to about 10×6 cm. and were of normal shape. The pancreas and adjacent glands were adherent, large, and hard. There were no metastases. Histologically the growth was more like an adenoma than a carcinoma, and was primary in the capsules. It was thus of quite a different character from the growths in the skin.

In Mr. Morris' case (2) the skin over the whole body contained scattered nodules the size of peas, but they could not be microscopically examined; he considered them metastases. It would seem that disease of the adrenals, which in some forms produces bronzing or discolouration, may in other cases be associated with hypertrophy of either the hair or the subcutaneous tissues, apart from metastases. The cause or significance of these rare complications is unknown, but Thornton's case would seem to show that they do not necessarily involve a bad prognosis.

As regards the mental defects in some of these cases, and the very marked apathy and weakness in nearly all, Alexander's researches on the adrenal as a source of lecithin, and the association of congenital cerebral and adrenal defects, are at any rate suggestive. Mr. Beadles (*v.*) gives it as his impression from experience at Colney Hatch that these bodies are particularly liable to hæmorrhage and degeneration among the insane.

The clinical course of the remaining Guy's cases will be referred to in Section B.

RENAL GROWTHS.

CONNECTIVE TISSUE GROWTHS.

The small fibromata dignified with the name of "nephritis interstitialis tuberosa", are considered inflammatory, and are in any case insignificant. Small white fatty masses are fairly often found imbedded in the kidney cortex; these may or may not be encapsuled, but in the fresh state are clearly defined by their colour. They

were formerly all classed as lipoma, and are indistinguishable by the naked eye, but under the microscope they divide themselves into lipomata, adrenal rests, and adenomata with fatty cells.

The lipomata arise in two ways:

I. By conversion of connective tissue into fat (Alsberg, Warthin, Beneke).

II. By inclusion of small particles of fat in the renal interlobular clefts, comparable to, and sometimes accompanied by, adrenal rests (Grawitz, Horn).

Calculous kidneys may undergo fatty change (Weir ii.), but these are not lipomata. Cases of true lipoma are described by Lazarus-Barlow, Parkes Weber (iii.), and others. Pure lipomata are rarely of clinical importance, though Alsberg removed a kidney enlarged to the size of a child's head by multiple lipomata, and Warthin removed one weighing 2 lbs.

The usual form of connective tissue tumour is a mixed one, composed of fibrous tissue, smooth muscle, fat, cartilage, etc., in varying proportions; these occur in the capsule and grow slowly. Busse describes one weighing nearly 20 lbs. which was of 25 years' duration.

Heyder has collected 18 cases operated on for this condition. None of them were diagnosed before operation. Ovarian cysts and hydatid were the commonest suggestions. Of these 9 were "successful," 8 died at once, and 1 in 5 months. He concludes that the symptoms are those of tumour without hæmaturia, or those signs of metastases which would be expected with a malignant growth of such size and duration. From the results of operation it seems clear that any attempt to remove them without a complete nephrectomy will fail. If they are left they may end by becoming sarcomatous. The fibrous tumour reported by Sir Samuel Wilks, and quoted by Busse and others, has been re-examined in the Guy's Museum and pronounced sarcomatous. In this case the patient had had hæmaturia and pain for ten years, and a tumour for six. He died of uræmia with no secondary deposits. This suggests that the growth was for a long time innocent.

Busse quotes a case of Tillman's, removal of a fibro-myxoma weighing 10 kgs., apparently innocent, but fatal from recurrence

within a year. In any case if such a growth were found it should clearly be removed with the kidney.

These are growths in which several kinds of connective tissue take part. They possibly originate in a confusion of embryonic elements, whence the variety of their structure; and in any case there seems to be no advantage in separating them into groups in which any one tissue predominates.

SARCOMA.

This group has been made to contain a good many diverse growths. The embryonic growths are commonly described as sarcoma, and the adrenal rest tumours as alveolar sarcoma. There are also cases of angeio-sarcoma, whose nature is still under dispute, and of lympho-sarcoma, which is said not to be a primary renal growth.

Freitag has attempted to study the residue of "ordinary" sarcoma which is left when these doubtful classes are removed, and finds them relatively rare; he does not attempt to arrive at a clinical type. He divides them into two forms—nodular and infiltrating. Of his three instances of the nodular form two are, unfortunately, inaccessible to me; the third, in a boy of eight (Van der Byl), is not a very good instance, as it appears to be embryonic, and no section was cut of it.

His own case, of the infiltrating form, presents very definite features. The patient was a man of 65. The disease ran an eight months' course without hæmaturia, with pain and weakness as the chief symptoms, and no tumour till shortly before death. Post mortem, there was no glandular invasion, and no metastases anywhere, except a nodule in the other kidney. On the other hand the growth had directly invaded two vertebræ, two ribs, the psoas and the posterior abdominal wall generally. It was a typical example of direct invasion as contrasted with blood or lymph diffusion. The growth microscopically consisted of every grade of cell, from a narrow spindle to a round cell—with a few muscle cells—but was mainly spindle cells. It could not be determined whether it started in the capsule or the kidney framework.

Freitag's division into nodular and infiltrating forms is probably

due to the description of these two forms of carcinoma, and is based on very few cases. Case 28 of the Guy's series is very definitely nodulated, but unless this feature is found to correspond to some structural or clinical peculiarity, as is the case with carcinoma, it seems unnecessary to insist on it. The significance of nodular forms of growth is discussed after carcinoma.

Of the Guy's cases, No. 28 has microscopically the purest sarcomatous structure. Except for an attack of pain 4 years ago the course of his illness extends over some three months; there were no urinary symptoms, and invasion was chiefly by the lymphatic system. No. 60 has marked local invasion. Other cases are Nos. 50, 45, 31, etc. No. 60 is a rapid invasion, 2 months; No. 50, 5 months; No. 45, about a year; and No. 31, 6 months.

If one could draw any conclusions from so few cases, it would be that the course of sarcoma is a rapid one, and since invasion takes place locally as well as by the usual channels, that it is not suited for operation. On the other hand one of Rovsing's cases was well 15 months after removal of a spindle-celled sarcoma, and the number of definite cases is not sufficient to generalize from. Those that begin as innocent mixed tumours, and reach a noticeable size before becoming malignant, can be removed in this stage. In these, as in other growths, everything depends on early exploration. Diagnosis without exposure is shown by Heyder's cases to be practically impossible, even when the growth is large; so that waiting is no great help in this respect, and increases the chances of malignancy.

ANGEIOMA AND ENDOTHELIOMA.

Small angeiomata such as occur in the liver are said to be found in the kidney—they are rare and insignificant. The only exception to their insignificance is when they are situated on a papilla, and give rise to continued hæmaturia. This condition is rare, and probably still more rarely recognized, it is possible that some cases of the so-called "essential" hæmaturia are of this kind. Mr. Hurry Fenwick (ii., 64) describes some cases of this condition. In one the hæmaturia had lasted for five years, and had been considered hysterical, the side affected was identified by the cystoscope,

the kidney appeared normal, but on incising the pelvis a single papilla was found covered with varicose vessels. Excision of this cured the bleeding. Mr. Fenwick records a precisely similar condition in another kidney, cured by excision of the papilla. In another case swabbing the papilla with perchloride of iron proved useless, and the kidney was removed at a second operation. In two other cases, assumed to be of this kind, incision and manipulation cured the hæmorrhage, without the pelvis being opened, and the explanation suggested is that the blood supply to the affected papilla was interfered with. This might explain some of the cases of "essential hæmaturia" which are reported cured by exploration. In a sixth case, in which hæmorrhage had been present for two years, an incision made with this intention gave no relief. The kidney was removed; and one papilla was found to have a capillary nævus, or angioma (pp. 74, 75). It is by no means clear whether this condition can fairly be called a "growth," but in view of these cases, it is of some clinical importance. Other vascular tumours have been described, but a vascular tumour is not necessarily an angioma. Rolleston and Kanthack describe a curious tumour with blood circulating in renal tubules, but they do not consider it a new growth. A curious vascular growth is described by Holmes, in which the patient had suffered from hæmaturia for two years, and, post mortem, the kidney (specimen 3591 in the R.C.S. museum) weighed 30 ozs., and had a spongy appearance with the spaces filled with soft vascular growth and clots. Pulsation and a bruit were noticed during life.

Numerous forms of growth have been described as originating in vessel walls—angiosarcoma (de Paoli Manasse Driessen), endothelioma (Manasse Hildebrand), perivascular sarcoma, venous and lymphatic endothelioma (Manasse). A certain number of these are probably identical with the adrenal rest tumours, and are considered so by those who describe them, or by others (Lubarsch). Others are apparently different, but no successful attempt has been made to lay down rules for distinguishing them (Lubarsch, Hansemann). Those that are identical with adrenal rest tumours may be classed under that head for purposes of study until the real nature of the growth is determined. The rest, which are not clinically very numerous or important, may be considered sarcoma. None of the

Guy's Hospital cases come under this heading, though in case 45 a concentric arrangement of spindle cells round the blood vessels is noted.

ADENOMA.

Adenomata occur as small round masses immediately under the capsule. They are sometimes encapsuled, often not ; and are usually, but not always, white and fatty. One form of pseudo-adenoma occurs in connection with granular kidneys, and is compared by Albarran to the proliferation of ducts which occurs in cirrhosis of the liver. Examples are described by Dr. Parkes Weber. Of Sudeck's 4 cases 3 occur in granular kidneys, but he states that they are not of this kind. Others occur (Ricker) in connection with scar tissue, apparently by the isolation of renal tubules. The genuine adenoma is probably a displacement of Wolffian or renal tubules during the formation of the gland. Two forms are described, tubular and cystic. The third form, the "alveolar adenoma" of Wechselbaum and Greenish, has been identified with adrenal rests. Instances of the tubular form are described by Ricker and Norman, of the cystic by Edmunds and Dalton. The latter usually has intracystic papillary growths, or trabeculæ. It is doubtful if they are really different, or merely stages of the same growth. At any rate the clinical importance of the latter form lies in its occasional malignancy, of which cases are described by Stanley Boyd and Kelynack. No case of this kind was recognized in the Guy's Hospital series, though innocent growths of both kinds are occasionally noted. A very striking case of this "malignant cyst adenoma" has been recently (1902) recorded by Voigt.

The patient, a woman of 38, underwent an abdominal hysterectomy for an enlargement of the uterus present 3 years. At the first dressing a tumour, hitherto masked, was noticed in the left loin. The patient made a good recovery, but died in the course of a few hours on the second day after she was allowed to leave her bed. The cause of death was pulmonary embolism. The left kidney, with the exception of the upper pole, was converted into a mass the size of a man's head, septate, adherent to the aorta, with one or two enlarged glands at the hilum. The adrenal was enlarged by growth. The

lumen of the vena cava was invaded by growth, and there were metastases in the liver. The tracheal, cervical and retroperitoneal glands, the lungs and pleura were also invaded. The uterus contained growth in the centre of four of the fibroids of the ordinary kind with which it was studded, and a mass was present in the right ovary. The tumours were all essentially papillary cysts, in places becoming carcinomatous. They were not the least like adrenal rest tumours, and were like papillary adenoma of the kidney. Voigt discusses whether they were primary in the kidney or uterus, or whether they were the simultaneous development of misplaced embryonic tubules. He decides that the growths in lungs and glands were metastases from the kidney growth by the blood and lymph stream respectively. The growths in the ovary and in the centre of the fibroids he thinks are from their character not the primary source, and from their position not metastases. He decides for the view that there has been a simultaneous development of rests of the primitive kidney in these three situations. This gains probability from the demonstration of what may be called the *adrenal rest tract*, i.e., a tract which extends from the under surface of the liver *via* adrenal, kidney, spermatic or ovarian vein, to ovaries, uterus, or testes. That this is a region along which embryonic misplacements are likely to occur is shown by the discovery at all points of it of adrenal rests, and at several points of the tumours which arise from them. If we imagine rests of the primitive kidney substituted for adrenal rests in this displacement (and "renal" rests in the adrenal have already been mentioned) there is nothing *a priori* impossible in the primary development of "renal" adenomata in any of these situations. The growth in the uterus is comparable to Mr. Eastwood's uterine adrenal rest tumour, that in the ovary to the rests recorded in this situation and in the testes by Ulrich and others. Their simultaneous development is again comparable to the condition described by Beneke as adrenal system disease. In the instance which he gives of this, there was present a simultaneous adenomatous growth of adrenal type in both adrenals and both kidneys—and a fair proportion of cases are found in which adenomata of the adrenal are bilateral. The fact that adrenal rest growths in the kidney are usually con-

geries of separate nodules suggests that where multiple rests exist several of them are likely to become active at the same time.

A case of Thoma's (vol i., p. 576) is of interest in comparison with Voigt's. In this there was an adenocystoma of the kidney in a woman aged 38, accompanied by a similar growth in the vaginal wall, and he considers them due to a simultaneous development of Wolffian rests. Thoma raises the question whether papilliferous cysts of the ovary and papillary cystadenoma of the kidney are not essentially the same, both originating in the remains of Wolffian tubules. This seems on the whole more reasonable than the other view, that they take origin from formed renal tubules.

As regards their occasional malignancy, in Mr. Stanley Boyd's case there seems to have been local recurrence. In Dr. Kelynack's (p. 119, *seq.*), there was a liver weighing 111 ozs., full of growth, a kidney the size of a child's head, with growth extending from it into the vena cava, a few nodules in the lungs, and a few small glands. The cysts were mostly filled with blood. Some excellent figures are given showing the intracystic papillomata, and the character of the deposits in the liver and the thrombus in the renal vein. In Voigt's case, where the metastases were even more extensive, it is noted that parts of the growth were becoming carcinomatous.

A corresponding "malignant tubular adenoma" has not been described; but in a case of Mr. Hurry Fenwick's (ii., 89), where the patient had had hæmaturia for $5\frac{1}{2}$ years from a small renal growth, Mr. Targett suggests, from the microscopical appearances, that it was originally an adenoma of the tubular type, which had lately assumed malignant characters. The nodular form of carcinoma has been attributed to malignant change in adenomata. If it is accepted that adenomata may develop simultaneously at several points of the adrenal rest tract, and also that they may become carcinomatous, it is not necessary to attribute to cystic or other adenoma any power of dissemination without change of type, and the term "malignant adenoma" may be rejected in favour of carcinoma, with considerable gain both in clearness and consistency.

That these growths are not malignant from the start is shewn by the frequent discovery of minute specimens post mortem, and by

the cases in which they have been successfully removed, even after they have attained some size. An encapsuled cystic adenoma, $2\frac{1}{2}$ inches across, was successfully removed by Dr. Edmunds from a girl aged 18, and a cystic tumour with papillary intracystic growth, weighing 2 lbs., was removed by Mr. Thomas Smith (sub. Willett) from a man of 19. The patient made a good recovery, hunted through the next season, and was reported well eighteen months later. Mr. Hurry Fenwick's case, mentioned above, was well 6 years later.

It would appear then that adenoma may arise either in the kidney or in any part of the "adrenal rest tract," and it may arise simultaneously in several situations. It is probably derived from remains of the Wolffian body. It may either remain minute, reach some size as an innocent tumour, or become malignant and destroy life. While it is still innocent it can be successfully removed.

The relation of this condition to cystic kidney, and simultaneous cystic disease of the liver and kidney, lies outside the scope of this paper.

CARCINOMA.

Just as the study of ordinary sarcoma has been neglected in favour of embryonic and other growths, or confused by their inclusion; so the ordinary renal carcinoma has been quite thrown into the shade by the enthusiastic interest taken in adrenal rest tumours.

Sudeck and Graupner, however, endeavour to treat of it by itself, and agree in distinguishing two forms, the nodular and the infiltrating. The naked eye distinction is that the nodular form consists of rounded masses of growth situated in a kidney otherwise practically normal, from which they are sharply defined; while in the infiltrating form the kidney as a whole is converted into growth by a diffuse malignant invasion.

In the first case we have a lobulated mass with, perhaps, half a normal kidney attached; in the second, a smooth homogeneous uniform mass of growth with no trace of kidney tissue. Sudeck distinguishes microscopically the little polymorphic cells of the infiltrating form from the cylindrical vesicular ones of the nodular form, which are often loaded with fat, surrounded by a delicate

stroma rich in vessels, and arranged in nodules separated by fibrous septa formed from compressed kidney tissue. He considers that those instances which are anything more than adenomata, are growths arising in adenomata. From his description it would seem that he includes in this class those growths which he calls renal adenomata, and other pathologists adrenal rests or adrenal rest tumours.

Graupner concludes that in the infiltrating form the character of the cells is that of pelvic rather than tubular epithelium, and that growth extends from the pelvis outwards along the urinary canals, while at the same time it projects inwards into the pelvis and causes hæmaturia. As for the nodular form, it is developed in the cortex, either from the convoluted tubules directly, or indirectly by malignant degeneration of renal adenomata.

A case of the infiltrating form is described by Dr. Beadles, and several have been reported in which the kidney is little, if at all, enlarged, but is wholly converted into growth.

Three successive cases of the nodular form are described by Terrillon.

As regards the clinical course, Israel, who gives examples of both, draws the following distinction. The nodular form, he says, remains long confined to the kidney (ii. 313), generally attains a considerable size, is easily palpable from its irregular shape, and can accordingly be recognized and treated relatively early in its course. The infiltrating form on the other hand does not alter the kidney for a long time, either in shape, colour, or consistence, and before any such change takes place it gives rise to metastases, so that diagnosis and treatment come too late. Clinically, then, the nodular form will fall among the cases of long duration which are operable, the infiltrating among those which are rapid and inoperable.

While these classes can be more or less distinguished amongst the cases from Guy's Hospital, the fact that the disease has run its entire course makes this much more difficult, for even the nodular form will ultimately destroy the whole kidney.

The most difficult distinction is between the nodular form of carcinoma arising in adenomata, and the nodular, cortical, slow-growing tumours which originate in adrenal rests. Sudeck over-

comes this by denying the existence of the latter. Another possible solution is to deny the existence of the former, but in all likelihood both occur. For clinical purposes there is no particular use in the distinction, since their course, according to Israel, is not very different.

NODULAR AND INFILTRATING GROWTHS GENERALLY.

The distinction between nodular and infiltrating growths, whether sarcoma, carcinoma, or any other variety, is probably to be found in their course rather than their kind. Some malignant growths are undoubtedly based on innocent growths which have been present for some time. Such a growth, whether connective tissue, gland tissue, or adrenal rest, will, in the course of its innocent development, form a defined encapsuled mass situated in an otherwise normal kidney. If such a mass then undergoes malignant change it will alter its structure and disseminate before its gross physical relations to the kidney have time to be materially changed. Its fibrous capsule is a very effective barrier for a time against infiltration of the rest of the gland. On the other hand, a growth which becomes malignant before it has developed such a barrier, or a growth of pelvic or renal epithelium which is malignant from the start, will be limited by kidney-capsule instead of growth capsule, and will invade the tissue within this area, *i.e.*, it will replace the renal tissue before deforming or enlarging the kidney as a mass. On this view the difference is not in the nature of the growth, the resultant sarcoma, or carcinoma, may be of precisely the same kind pathologically; but it is developed in a different relation to the fibrous barriers which condition its extension. If this is so, it is wrong to erect infiltrating and nodular forms of growth into real pathological kinds, although certain kinds of growth are from their life-history inclined to one or the other course. The real distinction is between growths primarily malignant, and innocent growths which subsequently become so. This corresponds with the clinical differences, and is practically most important. It is an essential distinction which is found in all the varieties of renal growth, and the division in the clinical section into cases of long and short duration is probably due to this, and not to the nature of the growths concerned.

PAPILLOMA.

Josselin de Jong has published (1904) a review of the 52 cases recorded up to date.

He found that these growths were liable to develop simultaneously in several places, *e.g.* pelvis, ureter, and bladder. Those that were anatomically benign passed on into carcinoma, and no single instance had occurred which was clinically benign, though on the strength of a microscopical examination many had been considered so till they recurred. The symptoms were those of kidney growth plus hydro- or hæmato-nephrosis. Diagnosis may be aided by finding fragments in the urine, or by seeing a growth at the opening of the ureter with the cystoscope. Treatment is in all cases nephrectomy and the excision of the ureter low down.

Drew's case illustrates these conclusions very well. He found a carcinoma of the kidney, a simple papilloma of the pelvis, and similar growths in the ureter and bladder which did not invade the wall. Metastases were present in the glands and tissues round. The villi were delicate and branching, with round or clubbed ends. He quotes eight cases, of which 4 were associated with calculi. One of these is Knowsley Thornton's, another is a specimen in the Guy's Hospital Museum.

Mr. Hurry Fenwick (i) has published a case which shows the importance of an extensive removal of the ureter.

The patient, a man of 70, had had hæmaturia for two years. With the cystoscope blood was found to issue from the right ureter. The kidney on this side was accordingly explored (by another surgeon), and nothing abnormal found. Two years after this, the patient came again, with a history that hæmaturia had been present since, and that a piece of villous growth had been found in the urine. Blood was again seen to issue from the right ureter. Accordingly the right kidney was removed and was found to have its pelvis full of villous growth, and its cortex honeycombed with cavities lined with the same growth. The patient recovered, but returned in a year's time with hæmaturia. Mr. Fenwick exposed the ureter, which contained a papillomatous tuft in its upper part, and excised it as far down as the point where it crossed the iliac vessels (both specimens are in the Royal College of Surgeons' Museum). There was a subsequent recurrence in the scar which was removed, and a deposit in the prostate. The patient died uræmic 5 years later.

Mr. Fenwick also mentions a case the converse of this, in which a papilloma was seen at the orifice of the right ureter, and removed, but a year later Knowsley Thornton was called on to excise the corresponding kidney for carcinoma.

These cases show that in this form of growth the pelvis and ureter must be considered together, and that a simple nephrectomy without excision of the ureter corresponds to partial excision of a carcinomatous organ. This is in accordance with their development (p. 221). At the same time, while growths of the pelvis almost always invade the kidney substance, growths of the renal substance practically never affect the ureter and bladder.

The first of these two cases shows that a papilloma may remain practically innocent for some time, though there is no doubt that it always becomes malignant eventually.

Another point illustrated by the first case is the difficulty of recognising this form of growth, even when the kidney is exposed. In specimen 3591A, of the Royal College of Surgeons' Museum, presented by the same surgeon, the kidney was found on exploration to be normal; but its pelvis "was distended with some soft material." On removal there was "a carcinoma of the pelvis, in places papilliferous."

Three cases of this kind have occurred at Guy's Hospital.

In case 27 most of the kidney was converted into growth, but there was a projecting mass springing from a point half way down the ureter, which suggests that it originated as a papilloma.

Case 62 had had hæmaturia for three years, and the pain, which came on in the last three months before admission, may have been due to the beginning of a glandular invasion. It is quite in accordance with De Jong's observations that, although the disease was far advanced, and glandular infection had occurred, the growth was microscopically innocent.

Case 64 illustrates the difficulty of recognising this condition at the time of operation. Fortunately this form of growth is one in which hæmaturia is constant, and the passage of fragments of growth most probable. Cystoscopy may also be of use, for it defines the side affected, and may show a secondary growth at the ureteral opening. Such a growth is in itself innocent and no contra-indication to operation.

ADRENAL RESTS.

At the time when Grawitz wrote his original paper (1883),

adrenal rests in the kidney, if observed at all, were described as lipomata. Since that time these structures have been discovered in a large proportion of bodies in various situations, and Grawitz' views on the origin of tumours from them (1884) have secured general acceptance.

Adrenal rests have been discovered in the liver, the neighbourhood of the adrenal, the cœliac plexus, the kidney and its immediate surroundings, the broad ligament, the spermatic cord and epididymis (Schmorl, Ulrich, Targett, Marchand and others). In fact it seems that they may occur at any point in a tract which begins in the liver, passes through the adrenals and kidneys, and then follows the spermatic or ovarian vein to its termination. Of this the upper part represents the region in which the adrenal is formed, the lower part the descent of the genital gland.

In the kidney these rests occur as little whitish subcapsular masses, which are by the naked eye indistinguishable from lipoma or adenoma, but under the microscope shew the characters of adrenal cortex and medulla (Targett Pitt), though they may contain cortex only (Ulrich), or a mass may be found consisting entirely of medulla (Eurich). The misplacement occurs in the form of inclusion of the whole or a main part (Ulrich) of the suprarenal within the renal capsule, or thin plates or multiple tiny nodules are found in the kidney at some distance from the normal adrenal. These rests are usually situated in the capsule, which encloses them and sends off a layer which separates them from the kidney. Sometimes separation is incomplete, and the two tissues interdigitate with nothing between them. (Ricker.)

The frequency with which adrenal rests are found is uncertain ; it varies of course with the care with which they are looked for. In the Guy's Hospital post-mortem records for 1892—1902, twelve are recorded, of which, however, only three were confirmed microscopically.

As they are minute and liable to occur at any point of a fairly long tract, a thorough search for them is never made in any ordinary necropsy. One successful method is to cut serial sections of the foetal broad ligament. Beneke quotes his colleague Schmorl as finding them in 92% of all bodies ; probably they occur much more

often than its usually believed. Clinically they are insignificant except as the starting point of adrenal rest tumours, and since they are common, and the tumours rare, their presence is not in itself important.

ADRENAL REST TUMOURS.

This name is perhaps the simplest of the many which have been applied to a class of tumours which begin with simple hypertrophy of an adrenal rest, go on to form a tumour which is in parts quite different from adrenal structure, and after a longer or shorter period of clinical innocence, disseminate and cause death. They have been, and are still, the subject of much controversy, ever since Grawitz published his paper in 1884, "On the origin of renal tumours from adrenal tissue."

The first dispute was between Sudeck, who denied their adrenal origin, and Grawitz, Horn, Lubarsch, and many others who affirmed it.

The second chief issue was raised by De Paoli, Driessen, Hanse-mann, and Hildebrand, who asserted that these growths were endotheliomata. It is possible to take the view that the cases reported are of several different kinds, that they are all adrenal, or all endotheliomata. Lubarsch and Hildebrand suggest that as the adrenal is the type of a perithelioma, any growth arising from it would be likely to have this character. Various side issues were raised about the presence of cysts and tubes in these growths, their glycogen contents, and other points. Reviews will be found in the papers of Gatti (1896), Kelly (1898) and in Borst's "*Geschwülstlehre*," (1902).

At the present time there is an almost universal agreement that such growths do occur and have an adrenal origin, but their frequency and essential character are both much disputed. They have been described alternately as carcinoma and sarcoma, either on the strength of divergent views of the origin of the adrenal body, which is not yet definitely settled, or on pure histological grounds, which are not much more reliable. It is probably not far from the truth to say that they look like carcinoma and behave like sarcoma.

The names, "*strumæ suprarenales aberratæ renis (event. malignæ)*,"

(Grawitz); "hypernephroma" (Burkhardt); "tumours of adrenal type" (Lubarsch); "adenocarcinoma," and others, all have disadvantages. Borst decides to call them provisionally "adrenal-tumours," and allots them an appendix to themselves.

The name used here is in the same way provisional, and begs the question of their origin, but it is impossible to denote this class of growths by any name whose connotation nobody will be found to dispute. The following description is taken largely from Borst for convenience. References to the original papers on which it is based will be found in the index.

The first stage of these growths is a simple hypertrophy of the adrenal rest. It conforms entirely to the type of normal adrenal cortex, *i.e.*, it consists of a fine capillary network in the meshes of which lie columns or masses of cells, with their bases applied directly to the capillary wall. The cells themselves are large, polygonal, epithelioid, with a large definite rounded nucleus and granular vacuolated protoplasm. They are always more or less infiltrated with fat, but are not degenerated, as is shown by the good staining of the nucleus. They contain glycogen and lecithin. When the fat is extracted the cells are left as large transparent vesicles, with scarcely any stainable protoplasm, but with a very definite limiting membrane, like that of a plant cell. (Fig. 1.)

The second stage is one of departure from the type in all directions, and a considerable increase of size of the mass as a whole. Instead of showing columns one or two cells broad, the cells form irregular masses or much thicker columns. The cells themselves become either smaller or larger, and some appear with many nuclei, or irregular or giant cells are found. The blood vessels become broad venous clefts. This may be called the stage of growth. (Fig. 2.)

The third stage—the stage of malignancy—is marked by a further departure from the type in some parts of the tumour. The cells become polymorphous, long or branched, lose their fatty contents, and vary in every possible way from the normal, with the result that some fields resemble carcinoma, others sarcoma, while in others the original structure is more or less preserved. In some cases there is hyaline degeneration of the vessels (Graupner). In some places

Growths of the Kidney and Adrenals.

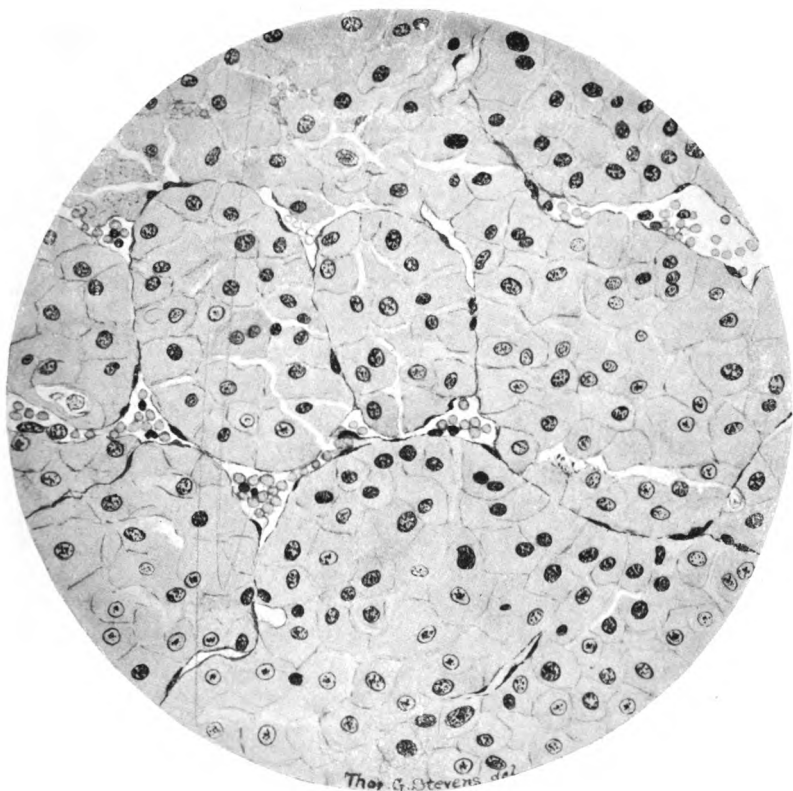


FIG. 1.

This is a portion of the growth represented in Fig. 3 \times 420. The field contains alveoli, bounded by capillaries, and here and there are seen the elongated nuclei of the single layer of endothelium which forms their walls. In places they are irregularly widened, and here blood corpuscles can be seen in the lumen; in the rest of their course they are visible as a thin dark line, marked by occasional nuclei. The cells which fill the alveoli are based directly on the capillary endothelium; they are large, polygonal, with large definite nuclei and nucleoli, and a fairly clear cell-body with a definite boundary. They are arranged in columns several cells broad, cut in different planes. In the recent state they were probably infiltrated with finely divided fat, but this has been extracted in the course of preparation. In one or two places blood can be seen extravasated between the cells.

Growths of the Kidney and Adrenals.

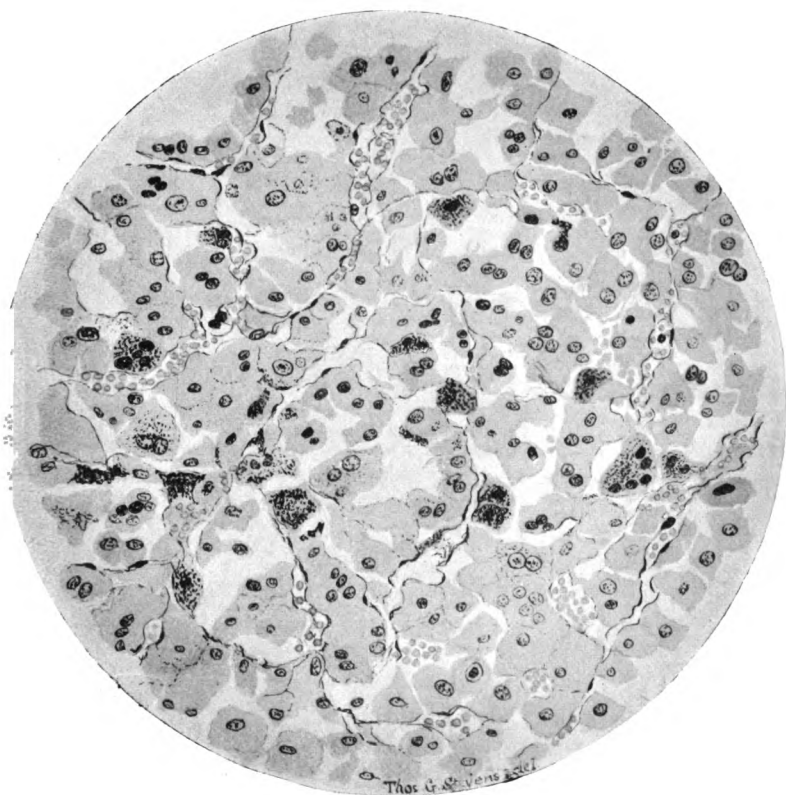


FIG. 2.

Another part of the same growth $\times 280$. In this the arrangement is rather less definite, and the cells less regular. The type of structure remains the same, but the alveoli are less obvious, the cells vary in size, and a number of anomalous cells are seen, containing dark granules, or two or more nuclei. One or two could fairly be described as giant cells, and one has five nuclei in its periphery.

Mr. Targett has been kind enough to examine this section, and thinks it may be best described as hypertrophy of an adrenal rest. It corresponds to the "stage of growth" described on p. 242. The character of the cells and their relation to the capillaries are the foundation for the terms "epithéliome aux cellules claires," and "endo-" or "perithelioma," which have been applied to adrenal rest growths.

papilliform masses of cells project into the lumen of the venous spaces.

The growth as a whole degenerates rapidly and hæmorrhage is apt to occur into it. The stages described above are not uniform all over the growth, and dissemination may occur at any time, so that a microscopical examination cannot decide whether the tumour is actually malignant or not. It is in any case potentially malignant.

Growth occurs from several centres simultaneously, as if the same stimulus had acted on several rests. The result is a collection of nodules lying immediately beneath the kidney capsule, and separated from one another, and from the renal substance, by fibrous septa formed from compressed kidney tissue. The origin of these septa is shewn by the occasional inclusion in them of degenerated renal tubules. Beyond them is a zone of nephritis shading off into healthy tissue. Macroscopically the nodules are originally white and fatty, but they soon break down centrally and their colours vary with the extent and date of hæmorrhage. The rest of the kidney remains for a long time normal.

These growths might *a priori* be expected at any point along the adrenal rest tract. Mr. Eastwood has supplied a link by finding one in the uterus, and he quotes one of Chiari's in the pelvis, but the commonest site is the kidney.

All sorts of criteria have been proposed for diagnosing these growths, but none of them are singly decisive. Great stress has been laid on the presence of glycogen; which is absent from the kidney, most kidney tumours, and the normal adrenal, but occurs in adrenal adenomata, adrenal rest tumours, and tumours of various kinds elsewhere (Lubarsch).

Mr. Targett suggests, as the cardinal features the distinct capsulation of the growth and its nodules; the characteristic arrangement of large cells in double columns without lumina, with their bases directly applied to the walls of the capillary network which takes the place of stroma; and the tendency to fatty infiltration and hæmorrhage.

Mr. Eastwood lays down that any tumour of which part exhibits a definite adrenal structure is of adrenal origin, and that others are not.

The presence of definite adrenal structure is accepted by all authorities as being necessary to prove an adrenal origin. Practically the difficulties seem to be considerable. While the presence of definite adrenal structure is good positive proof, the absence of it in the sections examined cannot always be held to prove a negative. In the more advanced stages whole regions of the growth may have a purely sarcomatous, or more commonly carcinomatous aspect, and while many ordinary carcinomata have very likely, as Mr. Targett suggests, been referred to this extraordinary class, it is probable that as many or more of these cases have been classed as ordinary carcinoma as a result of the examination of one section. Mr. McWeeney gives a figure of the "carcinomatous" part of his growth, which elsewhere showed undoubted adrenal tissue, and Beneke, Lubarsch, Burkhardt, Busse, Ulrich, and others have described similar fields. As Manasse puts it (p. 116) "To establish the identity of tumours arising from adrenal rests, we require the proof of adrenal tissue, or of the very characteristic adrenal-adenoma tissue. This will naturally not always be possible in tumours that have progressed far (*weit vorgeschrittenen*) and in such cases one is compelled to leave the precise histological diagnosis open" Burkhardt (p. 109) points out that the most typical parts are often destroyed by hæmorrhage, to which their structure renders them so liable, while adjacent atypical and less vascular parts survive.

In deciding the nature of a doubtful growth it is necessary to examine several different parts of it, and to consider the clinical and naked eye characters, as well as the metastases, for while these may vary widely from the original type (Askanazy), yet in some cases (Kelly), they may be more definitely adrenal than the primary growth. It is not suggested that the principle above stated should be relaxed, but only that in cases where the macroscopic characters, such as those mentioned by Mr. Targett, and the course of the case, are in favour of this origin, while the microscopic evidence is opposed or inconclusive (as in Maidlow's case), an open verdict should be returned.

As to the frequency with which they occur, there is no good evidence. Bayard Holmes asserts that they form one-third of all kidney tumours in adults. Kelly in 3098 necropsies found seven primary

renal growths, of which four were of this kind. Other authorities regard them as very rare. It is on the face of it unlikely that they occur so much more frequently in Germany as the comparison of the literature would suggest.

Several collections of these cases have been made. Lubarsch (1894) gives references to 29, including 3 described by de Paoli as angiosarcoma. Burkhardt (1900) collects 19 which have been operated on.

References to 41 cases will be found immediately after the bibliography. These exclude de Paoli's cases, but include two of Driessen's and one of Hildebrand's, which are published as endothelioma. The remainder are all published as adrenal rest tumours and possess some sort of clinical histories. Many others have been published in the form of museum specimens with no history, and cases found accidentally post mortem.

Rupprecht's case, which occurred in a child of $2\frac{1}{2}$, is also excluded.

Of these 41, 25 occurred in males, 16 in females.

The ages vary from 30 to 73, with an average of 50. Twenty-eight occur between 40 and 60, and five below 40.

A few cases have been recorded in which they were found in early life. Rupprecht's case was $2\frac{1}{2}$, and Bergstrand's 8. The kidney shown in Fig. 4, was considered from its size to be probably that of a child, but the specimen had no history attached to it. There is no reason why they should not occur in children, in fact from their origin they might be expected to, but apparently it is exceptional to find them below the age of 40.

Hæmaturia was present at some time in the course of the case in 26, and was the first sign in 20 out of 39. Burkhardt found it in 24 out of 30 cases. Three cases of exceptional length occur, one with a history of pain for 20 years, and two with tumour for 35 and 17 years respectively. In all of these hæmaturia was the second symptom, and marked the beginning of activity of the growth.

Excluding these, the average duration of symptoms is about $2\frac{1}{2}$ years.

In three cases the first symptoms were due to metastases in the lungs or bones. In 18 cases, pain, tumour, or both, was the first symptom.

The appearances of the growths accord with those described above.

A complete post-mortem examination was made in only 19 cases. In four of these there were no metastases, and in the successful operation cases we may assume there were none. Of the remaining 15, 11 shewed metastases exclusively by the blood stream. In one there was a general peritoneal infection, and in three there was, in addition to infection by the blood stream, some glandular invasion—but nothing in the least comparable to the cases of general glandular infection described below.

Clearly the typical invasion of these growths is entirely by the blood stream, *i.e.*, to lungs, bones and skin.

Twenty-eight of these cases were submitted to operation. Of these 10 died, eight from the operation, two from subsequent accidents; six died of recurrence or metastases in an average of 11 months; eleven went out well and were not further traced; and, four were well, one, one-and-a-half, six and seven years later respectively.

Some of the cases ran an apparently short course, but it must be considered that in the majority of them the course is only reckoned to the date of operation; in others the disease may have existed much longer than the symptoms. Some undoubtedly disseminated early and widely, but the striking feature of them as a group is the long duration of certain cases.

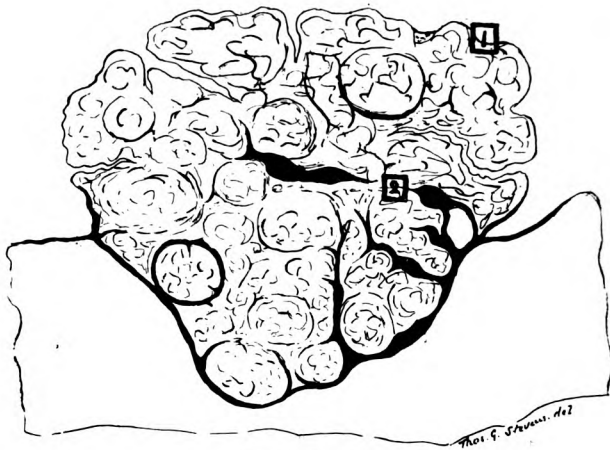
In Askanazy's case the patient had a tumour as a boy, and when he did his military training the belt used to give him pain by pressing on it. It then disappeared, and re-appeared at the age of 53, when it took on rapid growth, accompanied by hæmaturia, and ultimately killed him with extensive metastases.

Grawitz's case had hæmaturia for $5\frac{1}{2}$ years, and passed definite fragments of growth soon after the onset. In the two last years he was well enough to take his usual active summer holiday.

Busse reports a case of 6 years' hæmaturia, Manasse one of 5 years. Lubarsch gives one with slight pain for 6 years and hæmaturia for 3 years before operation. Hildebrand has one with 8 years intermittent hæmaturia, Perthes one with 5 years. Ulrich gives one with pain for 20 years. Driessen has one with 5 years, and Hansemann one with 17 years history of tumour.

Even if all these are not accepted at their face value, it is at least

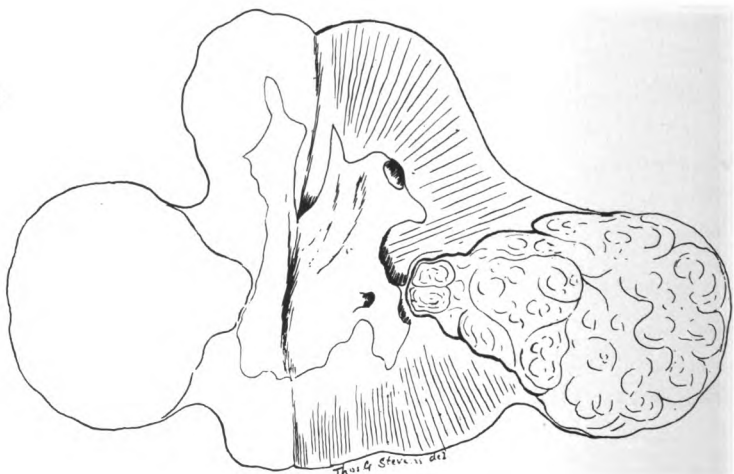
Growths of the Kidney and Adrenals.



Twice natural size

FIG. 3.

The renal tissue is left blank. The marks [1], [2]. show the position of the fields represented in figures 1 and 2.



Half natural size.

FIG 4.

remarkable that in this small collection of cases, ten, or about 25 % should have such long histories, and, taken in conjunction with the cases found post mortem (p. 261), it is clear that this form of growth may not only exist for some time as a clinically innocent tumour, but may allow of good general health and active life (as in Grawitz' case) for years after it has progressed far enough to give rise to hæmaturia.

There seems to be a general agreement as to the clinical characters of this group. Burkhardt lays stress on their long latency, which he puts at 4-5 years, their proneness to hæmaturia, and the size to which they attain without giving rise to metastases.

Lubarsch describes them as existing for years without symptoms, and being stirred into malignancy by illness or accident.

Perthes remarks on their duration, the slight effect they have on the general health, and the way in which their structure disposes them to hæmorrhage.

Mr. Bland Sutton (p. 129), states that "though these tumours are very vascular, and their central parts are often destroyed by extravasations of blood, they do not give rise to hæmaturia, because the tumour does not invade the renal pelvis. This is the most striking fact in their clinical history."

While this is perfectly true of adrenal rest tumours arising in the adrenal, it is impossible to agree with it as regards similar growths arising in the kidney. Originally cortical, they might be expected not to invade the pelvis, but apparently they often do, and other authorities seem to have found the frequency of hæmaturia, rather than its absence, the most striking fact in the clinical history.

Their method of development and extension is well illustrated by the two specimens shown in Figs. 3 and 4, of which one is in the museum of Guy's Hospital, and the other was obtained from the post-mortem room.

No representation is given of the ordinary adrenal rest, which is simply a minute subcortical nodule or plate. Fig. 3 shows a more advanced stage. The capsule has been stripped off, and the growth is left partly imbedded in the renal cortex, partly projecting from its surface as a mass like a cauliflower. The growth as a whole is separated from the renal tissue by a distinct fibrous capsule, formed

partly by a layer continuous with the kidney capsule and extending inwards from it, partly by a layer of compressed and atrophied kidney substance, which is wholly converted into fibrous tissue. Similar septa, continuous with this, extend into the growth itself, and divide it more or less perfectly into lobules. At the free edge some of these have been torn away with the kidney capsule from which they spring, leaving the surface irregularly nodular. The histology of this growth is illustrated by figures 1 and 2.

In the second specimen, (fig. 4), which is the one described by Mr. Targett (ii), a similar growth is shewn in a rather more advanced stage. The growth has not gone on to form a circular mass in the cortex as might have been expected, but has involved a segment of the kidney from cortex to pelvis, so that it forms a wedge, with its base projecting from the centre, its apex reaching the mucous membrane of the pelvis. As soon as this apex broke down the growth, which is by its structure prone to hæmorrhage, would have been in communication with the pelvis, and free hæmaturia would have followed from a relatively small and early growth. Several specimens have been described with this wedge-shaped extension, which illustrates one way in which early hæmaturia may be produced.

The cases which have been referred to this class amongst the Guy's records are only three—Cases 58, 69 and 70. From the fact that they all occur in the last four years, it is probable that a good many others occurred before, but were not recognized as belonging to this class. In none of these three cases is the evidence complete (p. 244).

Case 58 had hæmaturia as his chief symptom, without palpable tumour. His general health does not seem to have been very much affected, as he survived three operations, and ultimately died of gastric ulcer after the removal of the growth. When he died, nearly a year after the first hæmaturia, no metastases were found in the abdomen, and there is no obvious reason why the operation should not have been successful. In Case 69 a profuse hæmaturia was the chief symptom, together with pain caused by the clots in the bladder. There was some localized pain on the left side. Cystoscopy, with a view of localizing the source of the bleeding, would have been impossible; not because of the stricture, which offered less obstruction than the prostate, but on account of the quantity of blood and clot

in the bladder. (The case occurred during my house surgery). Post-mortem a small growth was found having the capsular relations described above, which had given rise to no metastases. Case 70 is one in which a growth had existed as a palpable tumour for a year, and was then apparently stimulated to growth by an attack of influenza, just as others appear to have been by accident or childbirth. A year later, with a large tumour, the patient's general health was still unaffected. The urine was normal and there was no hæmaturia. Operation was successful, and the patient, who is still in hospital, is doing well.

These cases illustrate very well the long duration without metastases, the early hæmaturia (in two cases out of three), the age incidence (all over 50), and the generally benign and operable characters of this form of growth. The first case died accidentally, the second was easily operable if there had been any indications for exploring the kidney, and the third, after certainly two years' duration, and probably much longer, seems in a fair way to recovery. The tumour in the last case is figured at the beginning of the paper, and a description accompanies the figures. (See Figs. 5 and 6.)

Summary.—A form of growth may arise from adrenal rests in the kidney which is distinguished microscopically by its subcapsular position, its encapsulation, its division into round nodules by fibrous septa, its white colour, and tendency to hæmorrhage and degeneration. Microscopically it presents in some parts typical adrenal tissue; in others a structure widely different. Its cells are often infiltrated with fat, without being degenerated.

Clinically it occurs between the ages of 40 and 60, and may exist for years with the symptoms of tumour or hæmaturia before it gives rise to metastases or affects the general health. In other cases it may disseminate early and widely, usually exclusively by the bloodstream.

EMBRYONIC TUMOURS.

This name (*embryonale geschwülste*) is a convenient one for the *sarcomata* occurring in children, which are essentially different from the adult growths. They are mixed growths (*mischgeschwülste*) arising from misplaced embryonic tissues, connective or glandular,

occurring sometimes before birth, usually in the first five years of life. Roughly most growths occurring under the age of twelve fall into this class.

It is not settled what is their exact origin. They have been referred to the Wolffian body (Birch-Hirschfeld, Targett), but Pick, in a review of the question, 1901, puts them back to an earlier stage, a displacement of undifferentiated mesoderm, while Mr. Shattock is inclined to think that they arise from the kidney.

They are composed of round cells, spindle cells, and gland tubules, in varying proportions. The glandular tissue is in parts regular, in parts converted into solid heaps of epithelial cells (adeno-carcinoma). In addition to these partially striated muscle tissue, cartilage, and fibrous tissue occur. Since different tumours, or different parts of the same tumour, may either contain any one of these constituents to the exclusion of the others, or any combination of them, they have been described by a corresponding variety of names. They are diagnosed mainly by the age at which they occur and their clinical characters. Much confusion has been caused by bracketing them with the various adult growths which they resemble microscopically.

They run a rapid course, generally growing from the hilum and expanding the kidney over them. The main symptom is tumour. Pain is variable, hæmaturia relatively rare, and the general health for some time unaffected. Metastasis occurs usually by the blood-stream but sometimes by the lymphatics. The secondary deposits are sarcomatous.

The most satisfactory account of their structure is that of Birch-Hirschfeld, and Walker gives a review.

In 1894 Mr. Targett took these growths as the subject of the Erasmus Wilson Lecture. He described and shewed specimens and lantern slides of the growths in the College of Surgeons, and other London museums. Several of these are reproduced in Dr. Kelynack's book. The classification was based on the primary anatomical relation of the growth to the kidney. An example was shewn of the round-celled perirenal growth which arises in the retroperitoneal tissues in children, and corresponds to the spindle celled and lipo-sarcomata of adults which are found in the same situation. The growth had originated at the hilum and had distorted

the kidney, it had only invaded the renal tissue at one small spot, but had compressed and invaded the ureter. These growths may reach a very considerable size without producing any other effect on the kidney than a deformity due to pressure.

The true renal growths were divided into subcapsular, hilar, and cortical. True subcapsular growth lying between the capsule and the kidney, as a subperiosteal sarcoma lies between the periosteum and bone, is rare, but may occur and reach a considerable size. The specimen shewn (3590 R.C.S.) was 8 inches in length, and was obtained from a girl aged 3; it was a round celled sarcoma with no glandular elements. Some growths were shewn occurring in the cortex and containing various cystic and glandular structures. The common form of growth is that starting in the hilum, which by its growth "unwraps" the kidney and expands its capsule. These tumours are in more than a third of the cases bilateral, as in the specimens shewn, and in the cases recorded by Abercrombie. Some of these contain fibrous tissue and striated muscle as well as adenoma.

The growths which are primary in the adrenal in childhood, were also considered. Anatomically they were found to present the following features:—

1. They produce marked flattening of the adjacent surface of the kidney, from which they are separated by a distinct layer of the renal capsule.

2. They push the kidney directly downwards, thereby rendering it unusually prominent, or sometimes the kidney becomes stretched like a cap over a part of the tumour.

3. By breaking through their capsule early in the course of the disease, the adjacent kidney is invaded and the size of the tumour is thereby materially enlarged. The liver and diaphragm likewise become speedily involved.

4. They are not bilateral like the renal tumours of childhood.

B.—CLINICAL COURSE.

INCIDENCE.

The total number of cases of primary growths of the kidney and adrenals which I have succeeded in finding in the Guy's Hospital records between 1826 and 1903 is 69, and of these 65 died in the

hospital. A total of 22·805 necropsies were performed during this period. This gives a proportion of one case in 350, or about ·28%.

The renal growths number 52, with a proportion of ·22%

Mr. Morris finds 5 in 2·610, or about ·2%.

Dr. Kelynak gives in two series, 9 in 4505, or ·2%, and 16 in 1400, or ·4%.

Clearly the frequency is about ·3% of both kinds together, and 2% of renal growths.

AGE.

It is generally stated that a large proportion of cases occur in the first five years of life, that the period from five to 30 is particularly free (Bland Sutton), and that after that date liability increases.

The Guy's cases are more or less in accordance with this, except in the absence of infantile cases, probably due to some point in hospital management.

They occur as follows :—

Five years and under	6 cases.
Between five and 25	3 "
„ 25 „ 30	4 "
„ 30 „ 40	12 "
„ 40 „ 50	10 "
„ 50 „ 60	21 "
„ 60 „ 70	14 "

The last class represents really a higher proportion than appears, as other causes of mortality have accounted for a good many people before the age of 60. So that from these cases it would seem that the main incidence began at 25 and increased rapidly after 50.

As regards sex, there were 57 males and 13 females: and, if the embryonic and adrenal cases are excluded, 49 males to 8 females. Walker and others find the sexes about equally affected in embryonic cases, so that the great difference here is probably partly accidental, partly due to the fact of the cases being adults. Dr. Kelynak quotes several authorities who find the male cases in excess. He himself analyses 135 cases and finds the females more numerous up to 40, the males at ages above 40 in the proportion of 27—19.

So that it is fair to say that the proportion of the sexes does not

differ much in the younger cases, but, with advancing age the male cases become much the more numerous. Growth occurred 34 times on the right side, 33 on the left.

CAUSATION.

The personal and family history of these cases is generally recorded in the original reports, though it has been omitted in the abstracts appended to this paper. As a rule it is good; certainly there is no illness or hereditary taint which occurs with constancy enough to have any claim to be discussed as a cause.

The two conditions which are usually considered as possible causes are calculus and injury.

As regards calculus, it may be primary, or secondary to the growth, and if it is primary it must occur with some frequency if it is to be considered a real cause.

Several cases have been reported in which there was reason to think the calculus was primary, as by Walsham and Newman (*c.f.* McCormac), and a large number in which the two conditions were associated. It would be difficult to estimate the proportion of cases showing this combination to those of growth without calculus, or calculus without growth, but probably it would be a small one.

The composition of the calculus is of importance, since a phosphatic one is more likely to be secondary to changes in the kidney. Rovsing quotes an interesting case where a small concretion passed was found to consist of calcium carbonate. The case was operated on for calculus, and a growth was discovered containing little centres of calcareous degeneration like the one passed. No calculus was present.

In the Guy's series, five cases are associated with stone, viz., 6, 27, 33, 56, and 57, and there are two preparations in the museum (1703, 1652), which illustrate the combination. Case 6 has no history.

Case 27 passed a calculus the size of a pea one year after the beginning of hæmaturia and three years before death. This is the case which had a papillomatous growth in the ureter, and we may infer (De Jong) that it was associated with a papilloma in the pelvis, and that the original growth was probably pelvic, and therefore quite possibly caused by the stone.

Case 33 has a small uric acid calculus at the top of the ureter. The growth arose "in the head of the kidney," sparing the lower end. It does not sound like a pelvic growth, and calculus can hardly be held responsible for growths elsewhere.

Case 56 is one with a history of 15 years hæmaturia, many calculi, a growth in the pelvis, and no metastases. In this case the calculi may quite well have been the cause, though they took some time to bring about the result.

Case 57, like the last, is accompanied with suppuration, and has a large branched calculus. Examination of the growth showed an epithelioma with squamous cells, and a "marked bird's nest arrangement." This points to the growth being pelvic.

So in three of these cases there is some evidence that the growth is pelvic in origin (whereas most growths are cortical), and associated with stone. In the other two there is merely the association. On the whole, it is probable that in some cases calculus is the cause of a malignant growth in the pelvis, though only a very small proportion of cases with calculus develop growth, and only a small proportion of renal growths are associated with calculus.

The question of injury is full of fallacies, since it is impossible to tell whether the growth was present before the injury, and how the injury affected the kidney.

Walker discusses the relation of injury to embryonic growths, and comes to the conclusion that it cannot be considered a real cause.

In the present series one of the embryonic cases (4) gives a history of a blow, and Case 68 (infantile adrenal growth) of a fall. Of the adults, 2, 16, 26, 32, 39, 41, 42, 55, 61, 67, have histories of some sort of injury. Cases 2, 16, 41, 55, have injuries preceding symptoms by periods from seven months downwards. In 26, symptoms came on a month after a severe labour, and in 70 after influenza.

Of the others, symptoms came on in 67 in a fortnight, in 61 at once, and in 32 in two days; while in 39 and 42, although active symptoms seemed to be induced by a fall in one case and a miscarriage in the other, yet tumour and pain had been noticed before this date.

It is possible for the healthy kidney to be injured by an accident,

but the accident has to be a severe one to reach it in its sheltered position. What probably happens in most of the cases is that growth is already present when the accident occurs. The period of latency, of some renal tumours at any rate, is a long one, and the cases which are found accidentally post mortem show that they may reach some size unnoticed. A growth is affected by injuries which would not hurt a normal kidney, and the injury either brings about hæmaturia, or determines the passage of the hitherto benign tumour into a malignant state. In 39 and 42, this was apparently the case, and in 67, 61, and 32, the interval is short for an entirely new growth to originate and give rise to symptoms.

Busse reports a case where a man fell carrying a sack, and immediately developed hæmaturia, which lasted till within six months of his death 6 years later. The injury was considered for purposes of compensation to be the cause of his illness. Post mortem, he was found to have an adrenal rest tumour, with deposits in the lungs. The fall cannot have originated an adrenal rest tumour, but it possibly determined its growth, or, at any rate, its bleeding.

So that it seems probable that while calculus in rare cases is a real cause of renal growth, injury is more likely to be the determining cause of the progress of a growth already present.

COURSE OF THE CASES.

In considering a case of growth of the kidney clinically there is no need to discuss whether it is innocent or malignant, for nearly all growths which can be recognized during life are malignant, and the few exceptions are very likely to become so.

The only practical question when once the diagnosis has been made, is whether local invasion and metastases have rendered operation useless or impossible. No treatment other than complete excision need be seriously considered. Coley's fluid has proved useless, and medical treatment is merely palliative.

As will be seen from the preceding section it is usually difficult to diagnose the nature of a growth post mortem; during life it is impossible. The most that can be done at present is to study the natural history of the cases, and endeavour to distinguish real

clinical kinds which may or may not be found later to coincide with the real kinds of histology.

The most important distinction for the surgeon is between those cases which remain long localized and operable, and those which become generalized at once. The distinction of next importance is between those which infect lymphatics and invade locally, and those which disseminate only by the blood stream. For in the second class there is the possibility of complete local removal and prolongation of life, even if unrecognized metastases are already present. In the first, anything like even a local removal is, for anatomical reasons, wholly out of the question.

The adrenal and embryonic cases will be first mentioned, and then the remaining cases considered *en bloc* from the clinical side.

ADRENAL GROWTHS.

The cases of adrenal growth in the Guy's series are 12 in number, of which two have already been referred to in the pathological section, 53 and 44. The others are 3, 15, 22, 30, 36, 40, 49, 59, 65, 68.

They fall into very distinct clinical types, both as regards their age, incidence and duration.

Three of them occur in children less than a year old, 65, 49, 30, and one in a child of $2\frac{1}{2}$ (68).

Six occur between the ages of 25 and 46, *i.e.*, in young adult life (cases 59, 40, 36, 22, 15, 3.)

Thus there is the same interval of immunity that occurs in growths of the kidney.

Again, as regards their duration, case 59 had pain for $4\frac{1}{2}$ years and tumour for a year. The duration of case 15 is uncertain. The remainder, including all the infantile cases, had a maximum duration of 8 months, and an average of 4 months.

Of the cases occurring in infancy, one (65) presented a tumour at birth and died in two weeks; another had rapid generalization in the skin and bones from the age of 2 months. The third is a case of a growth which gave rise to no symptoms, and the fourth had had tumour for a month, but shewed no other symptoms till a day or two before death.

Clearly nothing could have been done for these cases.

Of the rapidly fatal growths in adults, the symptoms of pain (in one case (40) going to the shoulder), weakness, vomiting, wasting, and tumour, with normal urine, occurred in different degrees. In one case (40) there were secondary deposits in the skin. In cases 22, 36 the growth spread through the diaphragm into the lungs.

None of them give the impression that anything could have been done for them.

Case 59 ran a wholly different course. Assuming the pain to have been due to the same cause throughout, the tumour had been in existence for $4\frac{1}{2}$ years, and it had been palpable for a year. Yet at the time of death the only metastases were "one or two nodules the size of a pea" in the lungs.

None of the viscera were affected, directly or indirectly. It is possible that operation at the time when the tumour was first discovered would have saved this patient. At the time when it was undertaken it was too late. But the operation would have been an exploratory one, for there were no localizing symptoms, and no mention is made of the weakness, vomiting, etc., which marked the other form of growth. As to its structure, the long duration and late diffusion by the blood stream are in accordance with what is known of adrenal rest tumours; the microscopic examination neither excludes or confirms this.

Of the rest, four were not examined, and five were small round-celled sarcoma. Case 40 is described as carcinoma. It has this in common with the case just described, that the glands are not infected.

Cases 40 and 15 are the only two adult cases in which both adrenals are the seat of growth, and these and 68 are the only cases in which the kidney is invaded. In case 15 the ovaries both contain growth in addition, this is either a case of "adrenal system disease," or retrograde infection by the ovarian veins.

Summary.—The Guy's Hospital cases of primary adrenal growth fall into two different classes.

1. A rapid growth occurring in 4 infants and 6 adults, spreading locally by extension, and diffusing by both lymph and blood channels, giving rise to weakness, vomiting, etc., and destroying life in an average time of 4 months—inoperable.

2. A slow growth occurring in one adult case, encapsuled, with no local extension, and late diffusion by the blood stream only; giving rise to no symptoms except tumour and pain, and not destroying life for $4\frac{1}{2}$ years from the first symptom, during part of which period it could have been successfully removed.

EMBRYONIC GROWTHS.

In the Guy's Hospital series only three cases of kidney growth occur which could be referred to this class, at 2, 11, and 5 years respectively. This is much below the usual proportion; Pick (quoted by Kelynack) states that 60% of all cases occur below five years of age, and Dr. Kelynack in his own series of 162 cases finds 84 or 52% below 10 years of age, of which 74 were under 5. Probably the explanation is that relatively few children were taken into this particular hospital.

Of these cases very little need be said. In case 12 the only symptom was a large tumour, which gave rise to no metastases. In cases 4 and 47, there is in each case a history of injury. In both, the growth extends into the vena cava and thence to the heart; in 47, the pulmonary artery is blocked by it, and the liver is enlarged to three times its normal size by deposits. In none was there hæmaturia, and only transient pain is noted.

These fully bear out the conclusions quoted above, that the chief symptom is tumour, usually without hæmaturia, and that metastasis occurs chiefly by the blood stream.

In case 12, as the tumour had already been noticed some months, it is possible that interference in an earlier stage with modern methods might have been successful. Case 4 came in moribund, and 47 was already rather advanced, for although signs of metastases were not present on his admission, his general health had failed for some time.

RENAL GROWTHS IN ADULTS.

CLASSIFICATION BY METASTASES.

There are several ways in which a renal growth may disseminate. The chief ones are the blood stream, the lymph channels, the ureter, and direct extension.

I.—Dissemination by the blood stream.

This probably occurs sooner or later in all these growths, it has been said to be a character of renal growths rather than of any particular kind of growth, so that for purposes of classification it is only important when it occurs independently of the other methods.

The usual way in which it occurs is that the growth comes to lie free in branches of the renal vein. It then extends to the adjacent vena cava, and either causes thrombosis of this vessel and extends upwards as a malignant thrombus towards the right auricle, as in cases 4, 55, 66, etc., or fragments are detached and form deposits in the lungs, more rarely in the bones of the cranium, ribs, muscles, and skin. Another way is for the growth to involve and penetrate some branch of the portal vein, giving rise to deposits in the liver.

In some cases the veins are thrombosed, the clot invaded, and a retrograde extension takes place. In case 19 thrombosis reached in this fashion down the iliac and femoral veins, and growth extended up the portal vein to the liver and down the mesenteric veins to their termination in the mucous membrane of the bowel, where it produced "heap-like elevations." A similar retrograde infection along the spermatic vein is reported and discussed by Sutter, in a case where the growth extends all down the cord to the testis.

Two classes of tumours which disseminate in this way almost exclusively are the embryonic and adrenal rest growths. In some cases the metastases are out of proportion to the primary growth, as in a case of Löwenhardt's, where a deposit in the clavicle had existed for four years and reached the size of a man's head without the primary renal growth making its presence known. In case 50 the only symptom was a deposit in the sternum.

II.—Dissemination by the Lymphatics.

Here again almost any growth will invade the adjacent glands at the hilum of the kidney after a certain time, probably by direct extension. Case 7 is an example of a blood extension with one or two glands at the hilum, and it is noted that the kidney "could be easily turned out" with them, so that they would not have been a bar to operation. Contrast with this case 43, a pure lymphatic

invasion, where the vena cava and lungs are normal, but the mesenteric portal, axillary, and inguinal glands are all infected, and case 54, in which the cervical glands are involved. Invasion of the liver may occur *via* the portal glands, and invasion of the pleura is noted in case 48 as occurring along the lymphatic channels.

It is clear that in a certain number of cases invasion takes place primarily by the lymph channels. It is necessary to include in this class cases where a blood infection has supervened late on an original lymph infection—as in case 10, in which lymph infection extends down to the inguinal glands and up to the mediastinal, but death was brought about by a shower of emboli in the lung, noticed at the post-mortem examination to be recent.

On the other hand, cases where there are only one or two glands in the neighbourhood of the kidney—as in Nos. 7, 12, 24, 39, 46—may be excluded from this class.

A good number of cases have invasion by both routes, and these fall into neither class, or rather into both. From the surgical point of view, both the lymphatic and the mixed invasion render local removal impossible; whereas a blood invasion, even with a gland or two at the hilum, does not. Of course, no operation would be undertaken if metastases were known to be present, but it is impossible in any case to be sure that there are none.

III.—*By the Ureter.*

This mode of extension is probably peculiar to the papillomata, and case 27 is the only instance among the Guy's cases. It is possibly a question of multiple primary growth rather than implantation. In the other forms of growth fragments are often passed down the ureter, but they never infect it.

IV.—*Direct Extension.*

This is not common in renal growths, and its rarity is due to the presence of a capsule. As suggested above, sarcoma developing in rather than within, the capsule, and growths of the adrenal which lie outside it, seem to have more marked powers of local extension. It is a bar to operation. It occurs in cases 6, 15, 22, 26, 27, 31, 34, 43, 52, 54, etc. In case 1 there is a direct invasion of the peritoneal cavity with peritonitis and multiple growths.

CASES OF INDEFINITE DURATION.

Growths found post mortem in patients who showed no symptoms during life.

Two of these cases are adrenal—49 and 53.

Of these, the latter is a mixed tumour already described, containing renal and adrenal tissue, and is essentially an adenoma. Its claim to inclusion depends on its size. The former is the size of a pea, and is described as a round-celled sarcoma; it occurred in a child of eight months, and is possibly the early stage of a malignant growth.

The remaining cases, 18, 21 and 38, occurred in adults, and are of considerable interest.

Case 18 is described in the "Transactions of the Pathological Society for 1875," vol. XXVII., p. 204. The other kidney was granular, and contained—apparently—adenomata. The growth in the affected kidney consisted of large round masses, and the cells, from their character and infiltration with fat, suggest an adrenal origin; though, as the case occurred 10 years before the publication of Grawitz's paper, this was not discussed while the specimen was fresh. There were no metastases, but the growth had in many places come to lie free in the veins, and there is no doubt that dissemination would ultimately have occurred by the blood stream (p. 259).

In cases 38 and 21 there were no secondary deposits. These growths were nodular, alveolated, marked off from the remaining kidney substance, and showed hæmorrhage into their substance. These characters and the transparency of the cells in 21 (*épithéliome aux cellules claires*), and the fact that the growths had reached some size without metastases, are all in accordance with what is known of adrenal rest tumours, but are not sufficient grounds for referring them to that class. At the same time it is noticeable that several of the tumours reported as having been found in this way have been adrenal rest tumours. Buday describes one in a man of 63, encapsuled, nodular, with big glassy cells and fatty infiltration. This resembled, in its position in the kidney, the one represented in Fig. 1. Driessen describes one the size of a child's head, found in a case of pneumonia (case III.). Ulrich quotes another

(case II.), and Grawitz describes one found in a woman who died of an operation for hernia, where the kidney was enlarged to three times its natural size by a growth which had given rise to no metastases. These four were all definitely adrenal rest growths. Dr. Hebb describes a growth in a man brought in moribund; but here the growth seems to have caused death by pulmonary embolism, though there were no metastases. The patient was walking in St. James' Park when he became faint, and was taken into hospital unconscious. The growth was a collection of nodules, and the description is not unlike an adrenal rest tumour. Mr. Beadles (i., ii., iii.) describes seven renal tumours found in lunatics; but it is not stated in all of them what symptoms, if any, occurred during life. In two there seem to have been none; in one hæmaturia referred to an accident; three of them showed no metastases. In the same way growths of the adrenal have been found accidentally; Mr. Wigglesworth describes one the size of an orange.

So that, excluding insignificant adenomata and minute growths of unknown character, of which many occur scattered through the Guy's records, it seems that in a fair number of instances a renal tumour has reached a considerable size without giving rise either to symptoms or metastases, and has been found accidentally on the death of a patient from some other cause. This is quite consistent with the cases mentioned elsewhere in which a tumour has been noticed for years before the appearance of any other sign, and with the long course of other cases. That the growths are not actively malignant for some time is shown by the rarity of metastases; that they ultimately become so is illustrated by the relation of the growth to the veins in case 18, and by the embolism in Dr. Hebb's case. In both of these it was a blood stream infection which finally occurred. Some of these cases have been seen to be adrenal rest tumours; others resemble them in various characters. It is probable that a large proportion of them are of this kind, but there are no grounds for asserting that they all are. Any growth which passed through a stage of innocence might be found in this way. Their significance as illustrating the long latency of some renal growths is independent of any guesses at their origin.

CASES CLASSIFIED BY DURATION OF SYMPTOMS.

The fallacy of this division is that the duration of symptoms is no index of the length of the disease. The cases which are found post mortem show this clearly. The occurrence of hæmaturia is an accident in the course of the growth; a tumour may be detected (Israel) when it is the size of a cherry, or it may remain impalpable even when its presence is presumed and its size considerable (case 58), while pain is a capricious symptom with many causes. Especially in cases in which a large mass is noticed for the first time on admission to hospital, we may presume that it has existed for a long time without symptoms.

Still this method of division has its advantages, since the duration of symptoms represents the longest interval before the natural end of the disease at which surgery can intervene, and the importance of early operation cannot be overrated: moreover since the error is usually in the same direction the results suffice for comparison.

We may exclude from consideration the cases of adrenal and embryonic growth, one or two cases discharged well, and five which were found accidentally post mortem. Of the 46 which remain the average duration of life was some two years. They are divided as follows:—

Under six months	18	} 22
Between 6 months and 1 year	4	
Between 1 year and 2 years	8	
Between 2 and 3 years	6	
Over 3 years	10	

Twenty-two, or nearly half, had a course of less than a year, while one was alive 12 years from the first symptoms, and one at the end of seven years had no metastases. They may be considered in two classes—30 of less than two years' duration, 16 of over two years.

SHORT CASES.

1, 2, 5, 8, 9, 10, 13, 16, 17, 20, 26, 28, 31, 32, 33, 40, 41, 42, 45, 48, 50, 52, 54, 55, 57, 58, 60, 66, 67, 69.

LONG CASES.

7, 19, 23, 24, 25, 27, 34, 35, 37, 39 43, 46, 51, 62, 63, 70.

SYMPTOMS.

Not much is to be gained by comparing the pain in these cases, as it is due to numerous causes (p. 267), and is very variable. Tumour was noticed in 15 of the short cases, and is not mentioned in 15. In the long cases it was present in 12, absent in 3. Hæmaturia was present in 13 of the short cases, absent in 17. In the long cases it was present in 12 cases, absent in 4. The metastases in the short cases were absent in 3, universal in 9; 7 were by the blood stream, 9 by the lymphatics. In those running a course under six months, they were universal in 8; by the blood stream in 4, and by the lymphatics in 5. In the long cases three are not available (62, 63, 70); of the rest, 2 were by both, 4 by neither, 1 by the lymphatics, and 6 by the blood stream. The only long case (43) in which dissemination was chiefly by the lymphatics, is also one of the minority as regards tumour and hæmaturia. Its claim to inclusion depends on a history of epigastric pain, worse after meals, extending over $2\frac{1}{2}$ years.

Converting these figures into percentages and comparing them, it is seen that in this small series of cases dissemination by both ways is commoner in the short cases, in the proportion of $2\frac{1}{2}$ to 1, and dissemination by the lymphatics, in the proportion of nearly 5 to 1. Dissemination by the blood stream, on the other hand, is commoner in the long cases in the proportion of 8 to 5. So that in these cases those of long duration extend by the blood stream rather than the lymphatics, those of short duration are relatively much more apt to extend by the lymphatics, or by both together.

As regards symptoms, a definite tumour is noted more commonly in the long cases in the proportion of 3 to 2. This is not important, it depends on the metastases in the short cases occurring early, before the tumour has time to become palpable, and occurring locally, so as to mask it.

Hæmaturia is relatively more common in the long cases, in the proportion of nine to five, and that this is not the result of their length is shown by the fact that it is the initial symptom in more than half of them.

The most remarkable, and clinically the most important, difference between these groups of cases is in their onset.

The long cases begin 8 with hæmaturia, 4 with tumour, 4 with pain.

Apart from these definite symptoms there is, as a rule, not much the matter with their general health for some time. Thus case 19 had had hæmaturia for 2 years at the time of his last admission, but he had been walking 10 miles daily for the last 9 months about his business, 24 boasted himself to have kept nearly continuously drunk for 3 years; 25, 35 and 70 were not troubled by their tumours, 37 went about with intermittent hæmaturia for 7 years, 46 bore children, 51 and 62 seem to have gone about in the intervals of hæmaturia, 63 went round various hospitals, and so on. So that roughly, at their onset, these cases presented the three classical signs, or some combination of them, and nothing else. Their general health was fairly good.

The beginning of the short cases is rather different. Eight (*i.e.* about $\frac{1}{3}$) have a definite hæmaturia, and three began with tumour. Eleven were markedly ill, and this was their first and main symptom, and of these, one had paralysis (9), and one (60) presented metastases in the deltoid and ribs. Of the others, three came in for œdema or ascites (55, 66, 48) and one for blockage of the ureter (52). Case 16 suffered solely from symptoms due to metastases in the brain, 40 had subcutaneous deposits before admission, and 50 came in for a secondary deposit in the sternum.

So that these cases, most of them, showed clinically a very different picture. In about a third of them hæmaturia and tumour were the first signs, in most of the others illness was the most marked feature, and in many of them other symptoms, due to metastases or interference with the big vessels, showed that the case was hopeless from the beginning.

The importance of this difference in the symptoms during the early stages is very great. The cases of embryonic tumour show (Bland Sutton p. 124) how little the presence of a renal tumour in itself affects the general health, and this is borne out in adults by the cases of long duration in this series. The "malignant cachexia" however, is produced as soon as direct extension or metastases affect any vital function, and in the kidney this is likely, for anatomical reasons, to occur soon. So that, apart from demonstrable metastases, any early failure of the general health, in excess of that due to pain or loss of blood, should raise the suspicion that the case

is one of the rapidly malignant group, and the same applies to any symptoms whatever, apart from pain, tumour, and hæmaturia.

Cases of long duration are by no means rare. A good many are recorded in the older literature. Of the more modern ones some have already been mentioned under the heading of adrenal rest tumours, and in a specimen of this kind, presented by Mr. Hurry Fenwick to the Royal College of Surgeons' Museum, (A3584B,) there was a history of hæmaturia extending over $3\frac{1}{2}$ years.

Of those that do not belong or have not been referred to this class, there is one on which Alm performed a successful nephrectomy which had had hæmaturia for 6 years and tumour for 3; and Kammerer reports a case of a woman who had had tumour for 6 years, with a recent rapid increase in size. With the cystoscope it was seen that no urine came from the affected side, and the colon was inflated and found to pass in front of the lower end of the mass. Nephrectomy was performed, and when the patient died a month later from an embolism, it was found that there were no metastases. Mr. Lunn reports a case with a history of 6 years' hæmaturia, a growth weighing $4\frac{1}{2}$ lbs. and projecting into the vena cava, and no metastases other than a small mass in the other kidney. Mr. Rushton Parker removed a renal tumour which had been noticed for more than seven years. The patient died of recurrence a month later. No doubt many other cases could be collected. When it is considered that the duration of symptoms is necessarily shorter, possibly many years shorter, than the duration of the disease, it is abundantly clear that some of these growths run a very slow course indeed.

Other cases may be fatal in a few weeks, as in Brault's case (11 weeks) and cases 22 (13 weeks), 26 (2-3 months), 52 (5 weeks), 60 (2 months). The difficulty is to distinguish cases of a growth which has been latent for some time and then takes on a rapidly malignant course, from those in which the growth is rapid from the start; but there can hardly be any doubt that the latter class exists.

SUMMARY.

From the clinical aspect both renal and adrenal growths in adults separate themselves into two classes.

1. Those which run a rapid course, invade locally, by the lymph channels, and the blood stream, present a variety of symptoms, and affect the general health almost from the first.

2. Those which exist and give rise to the classical symptoms of pain, tumour, and hæmaturia, or some of these, for a long time, often many years, before they cause any other symptoms or affect the general health. These growths are, as a rule, encapsuled and disseminate by the blood stream. When situated in the kidney they often cause an early hæmaturia; when in the adrenal, this symptom is absent, but they give the signs of other adrenal growths, and sometimes cause changes in the skin.

Histologically a considerable number of the latter class have been found, both in the kidney and adrenal, to have the structure of adrenal rest tumours. They also include the "nodular form" of carcinoma, and it is probable that this class includes any other cases in which, as in these, a benign growth precedes the malignant one.

The first class includes mixed celled sarcoma of the adrenals, and the infiltrating form of carcinoma. The first class will probably always be inoperable; the second are, owing to their symptoms, their method of extension, and their duration, capable of complete removal in their early stages.

DIAGNOSIS.

PAIN.

This is a very uncertain symptom, but occurs in all cases sooner or later, and may, as in Brault's case, be the chief or only sign. As a rule it is local, and radiates down to the groins, thighs or testes. In adrenal growths it is sometimes referred to the tip of the shoulder *via* the phrenic (Robson), and in renal growths tenderness is sometimes noted in the corresponding Head's area (cases 43, 51, 64).

Its causation is various; it may be due to direct pressure on a single nerve (case 8), or to involvement of the lumbar or even sacral nerves by extension or metastases. Gastric pain is caused by adhesions or involvement of the solar plexus; colicky pain is produced by the passage of blood clots, and vesical pain by their pressure on the bladder. Local pain depends usually on stretching or a local inflammation of the peritoneum; spinal pain on erosion of the vertebral bodies.

It is no safe index of the character or extent of the growth, for quite large tumours may run an almost painless course. At the same time it seems to be most severe in the more malignant forms.

The chief value of the symptom is that it may occur early and lead to a thorough examination. In itself it is merely an indication that something is wrong.

TUMOUR.

Tumours which have reached a size at which they give rise to œdema, venous obstruction, lung compression and the like, are easy enough to recognise. What is needed for successful treatment is their recognition while they are still very small.

Amongst the factors which render this difficult are obesity, ascites and tenderness. Further, only the lower end of the kidney is accessible, and most growths occur in the upper end. Enormous masses may be hidden away under the vault of the diaphragm, especially on the left side, and yet be impalpable (Rovsing, p. 414). Again, the infiltrating form of growth and the kind which invades locally are very difficult to feel compared to those which grow as nodular masses projecting from a normal kidney. The patients themselves very rarely take effective notice of a tumour till it has attained a fair size, unless it is accompanied by pain. By the time other symptoms drive them to seek advice the tumour is easy to recognize, and its recognition correspondingly useless.

At the same time palpation is very important, for precisely those growths (*viz.*, the nodular), which are easiest to appreciate, are also the most suitable for treatment. Israel lays great stress on palpation, chiefly on account of the success which has attended it in his hands. Others, less experienced or less fortunate, consider it relatively unimportant. In one case (p. 306), a patient, a man of 21, came to Israel with hæmaturia, and he was able to feel a lump the size of a 5pfennig piece in the anterior surface of the lower end of the kidney. This increased to the size of a cherry in the course of a month. The kidney was removed and a growth found extending from cortex to pelvis. The patient was well eleven years later (Heresco). Microscopically the growth was a medullary carcinoma.

In another case (p. 307), a girl of 6 had hæmaturia, and it was found that the blood came from the left ureter. The kidney was normal to palpation. Two months later it was found that the kidney was a little longer than the other, and had a diffuse smooth swelling on the middle third of its anterior and outer surfaces. Later a prominence like a distended pelvis was felt at the hilus. The kidney was accordingly exposed, and to careful palpation gave a sensation as if it had imbedded in it a harder mass the size of a hazel nut. On splitting it a medullary tumour was found at the depth of 4 m.m., extending along a pyramid and projecting as a rounded polypoid mass into the pelvis. Microscopically it was a sarcoma. The child was well 5 years and 10 months later (Heresco).

In yet another case, a man of 48, with hæmaturia, it was possible with great difficulty at the third attempt to feel on the lower end of the left kidney a prominence the size of a cherry. The kidney was excised; the growth proved to be a sarcoma, and the patient was well three years later (Heresco).

These are very brilliant results, but it must be remembered that it is only under certain conditions that a growth at this stage is palpable at all. For the growth must be of a certain kind, in a certain sort of patient, in a certain part of the organ, and according to Professor Israel must be sought for in a certain way, *i.e.*, with the patient lying on the sound side. One might fairly add that it must be sought for by some surgeon of Professor Israel's experience and capacity. Now these conditions are rarely combined; practically the case at this stage is usually in the hands of a man in general practice, who is not likely to make a large number of examinations, or to feel at the first attempt what Israel feels, "with great difficulty" at the third.

Moreover, tumour by itself is often misleading, Rovsing (case 7 of his series), tells how he explored the wrong side because the diseased kidney was impalpable, the sound one hypertrophied.

At the same time, Professor Israel's results are very good compared to those obtained by other surgeons, and his experience a large one. In the third paper referred to he summarizes his results at the time (1896). Of 17 cases of nephrectomy for growth, 2 had died at once, one a year later from acute peritonitis (no metastases), and three

were still under treatment. Of the remaining 11, 6 were well and free from recurrence at periods of 9, 7½, 5, 4, 3, and 1½ years respectively. These results he attributes to early diagnosis, rendered possible by palpation, and to convince the sceptical he made a drawing of the last-mentioned case (where the growth was the size of a cherry, situated two fingers' breadth from the lower end of the kidney), and demonstrated its accuracy at the operation.

It is noticeable, however, that in all these cases there was hæmaturia. This was the cause of the cases being examined in the first instance, and if it had been absent no amount of small prominences would have led to their exploration. Israel's advice that all surgeons should acquire the same skill in palpation is a counsel of perfection, but the lesson which these cases seem to convey is, that when there is an unexplained unilateral renal hæmaturia, the kidney should be carefully palpated, and then, whether a tumour is demonstrated or not, it should be explored. It is only in this way that the disease can be attacked sufficiently early—and early interference is essential to success.

HÆMATURIA AND URINARY CHANGES.

The value of hæmaturia can hardly be overestimated. Not only is it a frequent symptom, but it often occurs quite early. Morris quotes Denaclara as finding it as the first symptom in 68·8 per cent. of a series of 168 adult cases.

In the Guy's cases, at any rate, it occurred relatively most often and most early in the cases which were best suited for operation. Moreover, it is a symptom that the patient is prompt to notice and seek relief from. A man who would diagnose and treat his own pains, and not notice a fair-sized tumour, sends for his doctor in alarm at the first occurrence of a free hæmaturia. So that this symptom is one that can hardly be missed, and it occurs early in precisely those cases in which prompt treatment is most likely to be successful. At the same time it has many other causes, of which the one most often confused with growth is calculus. Bleeding from a growth is sudden, profuse, and independent of any apparent cause. It is not usually provoked by movement, or diminished by rest and dieting (42); sometimes, however, it follows an injury. At first blood

is nearly pure, and causes great pain by clotting; later, as it diminishes clots of the pelvis or ureter are often passed (51, 61, 64, 58). It continues for a few days, and then passes off altogether, usually to recur in a few weeks or months. In the interval the urine is normal, and commonly free from albumen, but blood corpuscles can often be demonstrated microscopically. After recurring several times, the bleeding may cease altogether, either from the blockage of the ureter or because the whole kidney is converted into growth and secretes no urine to wash the blood down. It is in the nodular vascular growths, such as adrenal rest tumours, where most of the kidney is functionally intact, that bleeding is most constant and persistent.

The first point to establish in a case of hæmaturia is whether the blood comes from the kidneys or bladder. The intimate mixture of blood and urine, the passage of ureteral clots, the possibility of getting fluid in the bladder clear enough to use the cystoscope, and the normal appearance of the bladder when this is done, are all important points. In case 58 this difficulty was only decided by cystotomy. If the bleeding is renal, the next question is which side it comes from. Generally, other symptoms are present to settle this, but if not, Heresco warmly recommends catheterization of the ureters. Latterly, a safer and simpler method has been introduced, in which various kinds of vertical partitions are introduced into the bladder, and urine from each side collected and examined separately. Mr. Lynn Thomas gives instances of its successful application. This has the additional advantage of allowing an estimation to be made of the urea excretion of the unaffected kidney without a separate catheterization, and is far easier to carry out. If an unilateral renal hæmaturia is found, and the diagnosis lies, as it often does, between stone and growth, the next step is to explore without waiting for further evidence. If the case is one of calculus there is no advantage in waiting; if it is one of growth, delay will probably be fatal. Mr. Hurry Fenwick says (ii. p. 99) "It should, I am sure, be a golden rule to explore every case of profuse renal hæmorrhage, and to be prepared for nephrectomy."

Apart from hæmaturia, a systematic examination of the urine is of great help in differential diagnosis. The presence of

gravel, tubercle bacilli or casts may clear up the case. The specific gravity is unaffected by growth, and, as a rule, the urea excretion is normal. An extreme instance is a case of Israel's, in which a woman dying with a carcinoma the size of a man's head excreted 33·7 gr. urea daily. Rovsing reports a case in which he abstained from operation on account of a deficient excretion of urea; the patient died of uræmia, and both kidneys were found to be involved in the growth. The specific gravity may be of value in excluding cystic kidney, a condition which is accompanied by tumour and intermittent hæmaturia.

A case of this condition associated towards the end with an adrenal growth is recorded by Dr. Lee Dickinson. The patient had suffered from tumour and hæmaturia for eight years. During the last six or seven months he showed progressive weakness and languor, and a dirty brown pigmentation appeared on the face, neck, and axillæ, with a few patches elsewhere. At the necropsy the kidney was found nearly entirely cystic, and the other kidney had hypertrophied and weighed 10 ozs. This accounted for the earlier symptoms; the later ones were due to a sarcoma of the adrenal with local invasion and secondary deposits.

An important positive sign which is rarely found is the passage of pieces of growth. Dr. Penrose records a case where tumour cells were passed, and post mortem a piece of growth the size of a nut lay loose in the bladder, while a similar mass grew from its neck. Rovsing found growth in the urine of cases 3, 6, and 7 of his series. It is noticed in some of the Guy's cases (23, 52, 58, etc.). In case 23 a distinct piece of villous growth was passed two years before death, imbedded in blood clot.

Hæmaturia is presumably caused by the breaking down of growth in the pelvis, so that it is during and after attacks of bleeding that fragments are most likely to occur. If the clots were examined and the urine centrifugalized on several occasions as a routine measure this sign would be more often obtained. Probably it occurs most often with the papillomata, but in case 23 the growth seems to have begun in the cortex. It is not possible to determine the earliest appearance of this sign, for as a rule it was not looked for, or the search not mentioned, before the cases came into hospital. It is only

owing to the circumstance of case 23 having been admitted twice that it is possible to assert that it occurred relatively early.

In a case of Grawitz's, an adrenal rest growth in the kidney, branching tufts ending in fine round knobs, and covered with a thick layer of large nucleated oval or irregular epithelial cells, were found in the urine $5\frac{1}{2}$ years before death, and practically at the beginning of the hæmaturia. Lubarsch mentions finding tumour cells in the urine (Case 7); and cells of various kinds are very often found, but they can never have the positive value of the tufts and fragments described above.

EXPLORATORY PUNCTURE.

This is warmly recommended by Israel, whose opinion is entitled to respect. In three cases he has extracted cells of growth, and even where this was wanting he has been able to satisfy himself that the tumour was solid, and to demonstrate, by the hæmaturia which followed the puncture, that it was renal.

On the other hand the nature of a growth cannot be diagnosed from a few cells, and the test is not infallible as regards the nature of the mass. For a needle may easily pass between the loculi of a pyonephrosis, or draw off fluid from a hydronephrosis dependent on growth.

The method was adopted in eight of the Guy's cases. In four cases blood or fluid was obtained, in two "rounded cells"; in one "gelatinous matter"; and in one nothing. The diagnosis was not materially forwarded, and of these eight cases two immediately developed acute suppurative peritonitis, and a third died five days later. Exploration through a lumbar incision would give vastly more information, and would hardly have a higher mortality. However cleanly the puncture may be done it is liable, in the absence of adhesions, to put a septic cavity in communication with the peritoneum (Case 29).

X RAYS.

These might be of great service in demonstrating the presence of a calculus where hæmaturia and pain are the chief symptoms, and the diagnosis lies between calculus and growth. Negative results

are not trustworthy, as Mr. Lucas has recently pointed out. Except for this purpose the rays have not been much used. One case (61) was skiagraphed with a negative result. Heresco gives an instance (157) where the tumour was shown in a radiograph lying under the diaphragm in a case where it was not palpable. This might be of value in some circumstances, and is always worth trying as a routine measure.

VARICOCELE.

Some stress has been laid on this symptom as indicating a glandular infection, and as contra-indicating operation. The frequency with which it occurs is unknown, as it is not marked when the patient is lying down, and the clinical reports are usually concerned with patients in bed. It has some significance as showing that there is interference with the return of blood along the spermatic vein, in cases where it has come on recently in a patient of middle age—beyond this it proves nothing. Mr. Morris found it with a tumour of the adrenal, and it may be dependent on pressure of the tumour on the veins, without metastases, or on thrombosis of the main trunk. As Heresco and others point out, varicocele may occur without glands, and glands without varicocele, and varicocele may disappear after a successful operation (Wallace), so that this symptom in itself cannot be held to contra-indicate operation.

LATE SYMPTOMS.

From a practical standpoint the numerous late symptoms which are described in detail in cases dying in the medical wards are of no importance, and the various ways in which the disease terminates may also be neglected. It is essential to make an early diagnosis, and it is almost equally essential to recognise when a case has become inoperable, but nothing is to be gained by studying the infinite variety of symptoms which are secondary to an enormous mass in the abdomen, or to metastases in the brain.

SIGNS THAT A CASE HAS BECOME INOPERABLE.

Instances have already been given in which the first symptoms are those due to metastases; such cases are inoperable from the

start. But in other cases it is often very difficult to decide whether any extension or metastases has occurred. The duration and size of the growth and the amount of pain and hæmaturia give little or no information on this point. Signs of mischief in the lungs and pleuræ, enlarged liver, paralyses, and œdema are among the commoner symptoms, but since there is no situation in which deposits may not occur, nothing short of a thorough general examination of the patient is of any use.

Perhaps the most important sign is failure of the general health. It has been pointed out in the "cases of long duration," (p. 265). and repeatedly observed in children, that a renal tumour in itself is quite compatible with the enjoyment of active life. It is usually only when the growth escapes from the capsule and invades surrounding structures, or gives rise to metastases in vital parts, that the condition called cachexia sets in. So that when a patient has had symptoms for some time, and then his health begins rapidly to fail, or where his illness is out of proportion to his pain or loss of blood, it is probably wise to abstain even from exploration. As Mr. Jacobson says, "When the history makes it probable that the growth has got beyond the earlier stage, when there is any extension to the lymphatic glands or other viscera, when there is nausea, emaciation, or a temperature inclined to fall, the time for operation has gone by." But supposing that none of these signs are present, and that an operation is taken in hand, it is still necessary to regard it as in the first instance an exploration, and to be prepared to desist if it becomes clear that no good can be done. Direct invasion of muscles, and a lymphatic invasion extending beyond the few glands situated actually on the hilum, are good reasons for stopping, while large tumours and extensive adhesions without invasion are merely mechanical difficulties which may or may not be superable.

An instance is case 41. Here there was a large tumour, pain, and a history of hæmaturia, with signs of mischief in the lungs, but these might well have been due to pressure of the mass. Otherwise the very extensive growth found post mortem seems to have given rise to few, if any, positive signs. At the same time the patient was noted to be pale and sallow, and he had been invalided for 19

weeks. Mr. Lucas explored, found evidence of glandular infection, and desisted; a course amply justified at the autopsy.

In a case of Abbé's, where he left behind two enlarged mesenteric glands, the child was well for four and a-half years, and then died of growth in the other kidney, which was probably entirely independent. But in an infant the mesenteric glands are not the usual route of invasion, nor is growth the only cause of their enlargement.

A case of Israel's has been quoted as shewing that a growth is in itself toxic by resorption. The case was an advanced one in a woman of 43, with hectic fever, vomiting, and signs of nephritis. On removal of the tumour all these signs disappeared, to reappear soon afterwards with the recurrence of the growth. But this was an inoperable case, in which the only object was to afford relief, metastases were presumably already present, and it is hardly fair to conclude from it that a growth in itself causes illness, when there are so many instances in which it does not.

So neither of these two cases can be said to controvert the principle that glandular infection and disproportionate illness contra-indicate operation.

EXPLORATION.

This is by far the most satisfactory means of diagnosis, and under modern conditions there is no reason why it should not be freely used, if the whole question is fairly and fully explained to the patient, and his consent obtained to any further operation which may be found necessary.

Rovsing in one case explored the suspected side, found it normal, and under the same anæsthetic explored the other, found a growth, and successfully removed it. This shows, if proof were necessary, that even a very thorough examination of a kidney does not do it any harm, for in this case the explored kidney had to do the whole work of the body at once. This patient had a recurrence in the scar four years later; this was excised, and he was well 5½ years from the first operation.

In exploration from behind the pelvis can be felt, calculi detected, the substance of the kidney palpated with a nicety im-

possible through the parietes, and what is more important still, the kidney can be seen. For most growths are cortical, and most growths are white, and their colour marks them out even before they become palpable. Of the Guy's cases all those that were operated on may be said to have been explored, in the sense that every operation is an exploration. But in case 58 the kidney was exposed in the absence of a palpable tumour, and in uncertainty as to the side affected, and the result fully justified this course. In case 51 two explorations were carried out, in the first the kidney was found rather large and hard, in the second, two months later, its anterior surface was found covered with white warty growth. On the first occasion the kidney was punctured and palpated. There is nothing in the report to show whether the anterior surface was examined or not. If it had been, it is possible that the colour of the growth would have led to its detection. The small amount of metastasis present post mortem suggests that its removal on the first occasion might have been successful. Another class of cases in which even exposure of the kidney leaves the diagnosis doubtful is that of the papillomata, where a fulness of the pelvis is in the early stages often the only sign. However, in the two cases of this kind a right conclusion was arrived at. It would seem to be essential to examine both surfaces of the kidney in a good light, to palpate the substance, and examine the pelvis before pronouncing it normal.

A large proportion of the recorded cases seem to have been submitted to the surgeon far too late. Descriptions of universal adhesions, networks of enlarged vessels, and tumours so large that they have to be delivered piecemeal, contrast in a very striking way with Israel's cases quoted above, where the growth formed a slight projection on an otherwise healthy kidney, and nephrectomy was correspondingly easy and successful. All surgeons are agreed that early diagnosis is the one thing essential to success, most allow that without exploration it is difficult, with it usually easy.

It is true that some large tumours have been successfully removed, but this is no justification for allowing any given tumour to become large. The course too often followed has been to watch

the case till unmistakable symptoms have settled at once the diagnosis and the prognosis, and then to send it to a hospital or a surgeon for operation. Compared with this method of dealing with malignant disease the purchase of the Sibylline books (which it closely resembles) was a prompt and business-like transaction.

If surgeons are to explore early they must be content to do so on the grounds of hæmaturia alone, or tumour alone—that is to say, hæmaturia or tumour of a certain kind, the hæmaturia apparently causeless, profuse, unilateral, renal, the tumour rounded, solid, slightly mobile, retrocolic. Israel's successful cases in most other surgeons' hands would probably have been cases of hæmaturia without tumour. Rovsing's successful case (well 5 years 4 months) had a kidney that could not be felt on the affected side. McWeeney's case (well 4 years) had no hæmaturia. Perthes' case (well 5 years) had no hæmaturia. Jordan's (Heresco, well 5½ years) had hæmaturia without tumour. Clearly these successes have been gained in many cases by exploring on the strength of one of these symptoms alone, and of the two, hæmaturia of the kind described above is the more significant. Of course, if exploration is freely used a certain number of kidneys will be exposed on wrong diagnoses. Of these none need be damaged, and many of those in which the bleeding is due to other causes can be cured. A few unnecessary incisions are a cheap price deliberately paid for early diagnosis in malignant cases.

Exploration in an early stage has been urged particularly by Kuster and Rovsing. The latter sums the whole matter up thus: (p. 450) "And so my belief is that if we would only keep the possibility of malignant growth before our eyes in every case of renal hæmaturia whose cause is not quite obvious, and promptly apply all our resources to determine the cause of the bleeding, that in future successful results of nephrectomy for growth would become considerably more numerous—and if only surgeons would undertake exploration more frequently and more boldly, but at the same time resign themselves to leave colossal adherent tumours wholly alone . . . I am sure that the mortality of this operation could be brought from its present considerable height almost to zero."

METHODS OF OPERATION.

As regards the two main methods, the anterior transperitoneal and the lumbar, there seems to be a general preference for the latter. The actual incision must be made to suit the individual case. The mortality of the anterior incision used to be about twice as great as that of the lumbar, partly from sepsis, partly because this method was used for very large growths, too big to be got out through the loin. This difference has now disappeared, and the mortality in adult cases is about the same. In children the anterior method is still the more fatal (Heresco). The peritoneum is likely to be opened in any case, but the posterior incision gives better access. The whole question is discussed in Mr. Jacobson's "Operations." Israel and others urge that the fat and glands should be cleared out in every case, just as the axilla is stripped in cancer of the breast. The cavity should then be pulled open with broad retractors, dried, and inspected with a good light; after which tears in the peritoneum are sewn up and the wound drained. If necessary clamp forceps may be left on the pedicle. Abbé's successful case (8 years) and Bloch's case, were instances of partial resection of the kidney, a proceeding which might be adopted in exceptionally suitable cases.

Apparently early interference in carefully selected cases is much more important than any points in the technique. In case 39 death occurred on the table from detachment of a malignant thrombus by manipulation; and a similar case is recorded by Kuster (I.). It is important to avoid rupture of the capsule if possible. In a case of Perthes' (F1.) this led to recurrence in the scar by contamination of the wound. Early interference would diminish both these special risks as well as those common to all operations.

RESULTS OF OPERATION.

A.—ADRENAL GROWTHS.

Not very many operations have been done for this condition, and in no case has it been diagnosed beforehand.

Mr. Morris has published three cases, and Mr. Mayo Robson three, with reference to nine others—most of them proved fatal soon after operation

In the case of Knowsley Thornton's already mentioned (p. 225), operation brought about a great improvement in the patient's health and allowed her to resume active life. Unfortunately she died of recurrence two years later, but this result may be considered satisfactory, and as the tumour weighed 20 lbs. at the time of operation the case may be considered one in which operation came too late, rather than one which was inoperable.

Mr. Mayo Robson removed an adrenal tumour the size of an orange together with a wedge from the kidney, and the patient was known to be well nearly two years later.

Curtis (1900) showed a woman, aged 49, from whom he had removed an adrenal growth a year and a half before. In this case the urine was practically normal; the symptoms were dyspepsia and discomfort, and the chief positive sign was tumour. The growth partly involved the kidney, and the two together weighed 2 lbs. 2 ozs., and measured 16 × 17 inches.

In Mr. Mayo Robson's successful case the growth was an adrenal rest tumour, in Knowsley Thornton's case the description of the specimen in the catalogue of the Royal College of Surgeons' Museum suggests that it was of the same kind, and in Curtis's no details are given. It is known that these growths occur and reach some size in the adrenal (p. 222), and it is probable that the operable cases of growth will be found to be mostly of this kind.

In case 59 there was a history of four years' pain and a year's tumour with scarcely any metastases, so that probably early exploration with no diagnosis, or a wrong one, would have saved the patient; at the time when she reached the hospital it was too late. So that although the infantile adrenal growths, and the majority of those in adults, are likely to remain inoperable, the cases quoted above show that in a certain number early operation has a good chance of success. In these, even more than in renal growths, it is necessary to explore early, for hæmaturia practically never occurs, the tumour is often not retrocolic, and the characteristic symptoms are vague and late. In the case of growths that reach a considerable size under the vault of the diaphragm on the left side, it is possible that skiagraphy may be of some use, but as a general rule it is unlikely that a correct diagnosis will ever be made without an incision.

RENAL GROWTHS.

I.—IMMEDIATE RESULTS.

Heresco finds that the immediate mortality, *i.e.*, the proportion of cases dying within a month of the operation, was about 60 per cent. before 1890, and has since been reduced to less than 20 per cent. In his 53 cases in infants he found a mortality of 17 per cent. as against one of 60 per cent. in cases occurring before 1890. Probably this figure is too low, as Döderlein in a series of 47 cases (1894) finds it 40 per cent., and Walker in 74 cases finds it 36 per cent.

Of the nine completed nephrectomies in the Guy's series one died of gastric ulcer, two died at once, and one in two days. The five others, which are the five most recent, all survived the operation over a month, three were sent out well, one transferred to an infirmary, and one is still in the hospital. This fully agrees with the improvement in immediate results found by Heresco.

Apart from sepsis and a wrong selection of cases there is a high immediate mortality connected with nephrectomy. This is due to various causes, but one on which Israel is inclined to lay stress is a nephritis of the other kidney from chloroform. Rindskopf has made observations on this point, and finds that it occurs in a slight form in about one-third of all cases operated on under this anæsthetic, and in 82 per cent. of operations on the kidneys. It is shown by the passage of casts, albumen, etc., coming on a day or two after operation and lasting two or three days. In most cases it passes unnoticed, and in fatal cases it can only be detected by the microscope and is consequently usually missed. In case 6 of his series Israel satisfied himself that this was the cause of death. In case 42 of the Guy's series death followed two days after the operation with vomiting. It is noted that chloroform was administered for nearly two hours. Post mortem the remaining kidney was noticed to be "anæmic." Possibly death was due to this cause. The question of chloroform intoxication as a whole is discussed by Kocher (*Operations-lehre*, p. 15 *seq.*). The reason of its special importance in these cases is that it affects a kidney on which an exceptional strain is suddenly put.

Whatever may have been the causes of death in the older cases

it is clear that the immediate mortality has been much reduced by modern methods, and is now about 20 per cent.

II.—REMOTE RESULTS.

ADULTS.

It is difficult to draw any safe conclusions as to the proportion of cures from any series of cases collected from the literature. The fact of publication generally involves selection, apart from any that may occur in the compilation of a series, and the after-history is incomplete in most. Heresco has written for the after-histories of a large number of published cases, and his results are consequently much fuller than any to be extracted from the rest of the literature, but his conclusions are subject to the limitations suggested above. Of his 112 adult cases 89 survived the operation, and he obtained an after-history in 62 of them. Of the cases seen after three years 10 were well at periods up to 7 years. One died without recurrence at 4 years 9 months, and one died of recurrence in $3\frac{1}{2}$ years. So that 11, or 10 per cent. were "cured" in the sense that they were known to be well at the end of three years and are not known to have had recurrence since. Fifteen others were well at the end of a year. One of these then died from another cause. Three had recurrence between one and three years. In all 22 are known to have had recurrence, and in 27 there is no history. So it is established beyond doubt that of this series of cases some proportion over 10 per cent. were "cured" by operation in the sense defined above, and a number of others were well at the end of a year.

Heresco classifies the results according to the form of growth, but this is very unsafe ground, for reasons already stated. The operative results of the adrenal rest tumours have already been mentioned separately.

CHILDREN.

Heresco has 56 infantile cases, and an after-history in 24. Of these 16 died of recurrence, 8 were alive at periods varying from 9 months to 6 years, 4 being over 3 years.

Walker collected 74 cases, and found that 27 died of the operation, 28 had recurrence, 15 were lost sight of, and 4 survived over 3 years.

Since the publication of his paper one of these cases (Abbé) has died of recurrence after $4\frac{3}{4}$ years, and one of his incomplete cases (Malcolm) has since been reported well at the end of 10 years, and another (Döderlein) at the end of 6. He found 5·4 per cent. of cures, and an average duration of 16 months of life in the operated cases, as opposed to 8 months in those unoperated. These alterations would bring the proportion of cures to 6·7 per cent.

The following are the successful cases up to date :—

SUCCESSFUL CASES OF NEPHRECTOMY FOR GROWTH IN CHILDREN.

SURVIVAL OVER 3 YEARS.

NO SUBSEQUENT RECURRENCE REPORTED.

Surgeon.	Age and Sex of Child	Reference to Operation.	Reference to Survival.	Time of Survival.
Malcolm	Girl, $1\frac{1}{2}$ yrs.	Trans. Clin. Soc. XXVII. 94	Letter to Mr. Bland Sutton. Tumours, innocent and malignant. 1903. p. 126	10 yrs.
Abbé	Girl, $1\frac{3}{4}$ yrs.	Annals of Surgery XIX. 58	Annals of Surgery XXXI. 760	8 yrs.
Döderlein	Girl, 7 yrs.	Cbl. f. d. Path. der Harn-Organ V. 5	Letter from Perthes to Heresco. p. 89	6 yrs.
Israel	Girl, 6 yrs.	Lang. Archiv. XLVII. 307	Letter to Heresco. p. 124	$5\frac{1}{2}$ yrs.
Israel	Boy, 14 yrs.	" " p. 322.438	Ibidem	5 yrs.
Schmid	Boy. $\frac{6}{12}$ yr.	Münch Med. Woch. XXXIX. 256	Cbl. f. Chirurgie XX. 672	3 yrs.
Abbott	Girl, 1 yr.	Letter from Mr. Abbott.	Child seen well Dec., 1904. O.R.	$4\frac{1}{2}$ yrs.

These figures are an understatement of the amount of success obtained, for so many cases are lost sight of, and it has been seen that at least two of Walker's "cases with incomplete history" have since turned out to be cures.

As regards Abbé's case of recurrence after $4\frac{3}{4}$ years, the second growth occurred rapidly, at the end of a considerable interval, in the

other kidney—it is not likely to be an actual recurrence, but rather a fresh development—and if this view is taken, the case is really one of “death from intercurrent affection.”

Mr. Abbott proposes to publish his case in full at a later date, but in the meanwhile has been so kind as to furnish me with the following particulars, and to give me permission to include them.

Nellie A., aged 1, was admitted to the Evelina Hospital with growth of the right kidney. The operation was performed on March 29th, 1900. A long oblique incision was used, opening the peritoneum in its anterior part. Great difficulty was experienced during part of the operation. The child did well afterwards. The tumour was thought by Mr. Targett to be a columnar-celled carcinoma; it was mixed in structure, and Mr. Shattock was inclined to think it a sarcoma. On May 6th, 1902, the child was re-admitted to St. Thomas' for what was considered to be recurrence in the abdominal wall and glands. A large piece of abdominal wall and muscles was removed right down to the iliac crest, also some lumbar glands. These and the wall were dense, hard and fibrous, and contained a fluid that looked like pus in numerous loculated spaces. There had never been any temperature. No cultivation grew from this fluid, and no trace of growth was found in the parts removed. The child made a good recovery, though it had a large hole in its abdominal wall, which was much weakened.

The child was last seen on December 7th, 1904. There is no sign of recurrence; the scar is flush with the skin unless the child coughs or cries, when a small hernia appears, chiefly at the upper end, this is easily controlled by a belt and pad; the opposite kidney is very readily palpable, and is enlarged, presumably by a compensatory hypertrophy.

A good many other successful adult cases might no doubt be collected from the literature, or by writing to the surgeons. For example, two are mentioned in Heresco's preface, one of Krönlein (well 13 years) and one of Israel's (well 11 years). Fenger reports one well 7½ years, and Knowsley Thornton one well 7 years. In the last-mentioned the tumour weighed 11 lbs., had been present for six years, and was said to resemble adrenal tissue. Probably it was an adrenal rest growth. Maidlow's case was well 3 years later (Bland Sutton, p. 131); in this case the growth weighed 7 lbs. But a few cases more or less make no difference to the conclusion which the recorded cases fully establish, that in some cases of renal growth a permanent cure can be obtained by operation. Although the successes are not very numerous, it must be remembered that they

are all relatively recent. Heresco had difficulty, he says (p. 30) in finding two or three cases before 1890 which had survived operation for more than a year. There is no difficulty now in finding a number that have survived more than three.

The difference that has been brought about in the outlook is well seen by comparing the two last editions of Mr. Butlin's *Operative Treatment of Malignant Disease*. In 1887 all operation is practically condemned. In 1900, Mr. Bruce Clarke is able to write about this operation: "There are reasons for hoping that better results may be obtained from it in the immediate future. The mortality due to the operation is already very much lower than it was, and is likely to be still further diminished by careful attention to the diagnosis of the disease in the earlier stages."

The infantile cases are less promising than the adults. Not only does the disease sometimes begin before birth, but hæmaturia is relatively rare, and the diagnosis is generally made when a large tumour is already present; while infants are not in any case good subjects for extensive abdominal operations. The selection of suitable cases is also extremely difficult. Abbé was unable to state any points that distinguished his successful cases from the unsuccessful at the time of operation. Since, however, the disease is inevitably fatal, and there is a slight chance of success, there can be no doubt that operation in suitable cases is justified.

In adult cases there is no encouragement in the results quoted above for interference in all cases or at all stages. Probably, as in children, the majority of cases will remain inoperable. What can reasonably be expected is that early diagnosis will raise the total of successes, and careful selection increase their proportion to the number of nephrectomies performed.

SUMMARY.

Our knowledge of the different forms of growth is increasing, but at present it is impossible to diagnose them during life, and their separation after death presents peculiar difficulties. Clinically both renal and adrenal growths fall into two classes, one, rapid, diffuse, disseminating early and affecting the general health; the other, slow, encapsuled, disseminating late and usually by the

blood stream, and giving rise to pain, tumour, or hæmaturia for some time without other symptoms. The essential difference between these classes is probably that the latter consists of those growths (whatever their structure) which exist as innocent tumours for some time before they become malignant, whereas the former are malignant from the start. Since in certain kinds of tumours, such as the adrenal rest growths, a benign stage is the rule, whereas in others, such as pelvic carcinoma, it is the exception, it is natural that the majority should impress their secondary characters on the class as a whole. Consequently we find the latter class showing the same preference for dissemination by the blood stream that is shewn by the adrenal rest growths, but less absolutely.

Since carcinoma, epithelioma, sarcoma, and malignant adrenal rest growths may any of them be preceded for variable periods by adenoma, papilloma, connective tissue growths, and innocent adrenal rest growths respectively it will probably never be possible to connect the structure of a growth absolutely with its clinical course. At the same time there are grounds for thinking that growths of an adrenal type are relatively more likely to run a slow and favourable course.

The diagnosis of the presence of a growth may be much helped by the employment, in addition to the usual methods of examination, of the cystoscope and urine separator, by a repeated examination of the urine and the deposits obtained by centrifugalizing it, and by the X-rays. Diagnosis is often impossible without exploration, and if suspicion of a growth is aroused by any single symptom, of which hæmaturia of a certain kind is the most important, this should be employed without delay. No hæmaturia should ever be left unaccounted for, and as few tumours as possible. Disproportionate failure of the general health and evidence of metastases forbid operation; varicocele, skin changes, and tumours of a moderate size do not. The immediate mortality of the operation has been much reduced since 1890, and is now about 20 per cent. Before that date scarcely any successes were recorded; since then it has been found possible, in cases taken early, to bring about a fair number of complete cures extending over three years or more. The whole question turns on early diagnosis, and much better results

may confidently be expected if adults in whom there is any suspicion of this disease are at once submitted to a thorough examination, including, if necessary, exploration through a lumbar incision. It is unlikely that a corresponding improvement will be obtained in the growths that occur in children, but the results justify operation in carefully selected cases.

In conclusion, I should like to express my thanks to those members of the Staff who have kindly allowed me to use their cases, and particularly to Mr. Jacobson, to whose suggestion and encouragement this paper owes its existence.

ADRENAL REST TUMOURS.

References will be found in the index under the following names:—Grawitz, Johnson, Israel (2), Busse (4), Askanazy, Beneke, Gatti, Manasse, Burkhardt (4), Lubarsch (5), Kelly (2), Hildebrand (3, "endotheliomata"), Holmes, Perthes (3), Boyd, Ulrich (3), Graupner, Fairbairn, McWeeney (2), Driessen (2, "endotheliomata"), Hanseemann, Löwenhardt. These all occur in the kidney, and have some kind of clinical history.

For other instances of this form of growth, see under Rupprecht, De Paoli, Bergstrand, Bland Sutton (p. 130), Eastwood, Kuster, Targett, Thelwall Thomas, Knowsley Thornton, and the authors mentioned above, also the cases figured, and those included in the Guy's series, 58, 69, 70.

BIBLIOGRAPHY.

-
- ABBÉ. I. 1894. *Annals of Surgery*, XIX., 58. Three nephrectomies in children, one partial.
- II. " " " " 92. Discussion.
- III. 1895. " " XXII., 269. Discussion, cases shown.
- IV. 1897. " " XXV., 360. Recurrence in one case.
- V. 1898. " " XXX. 317. A nephrectomy.
- VI. 1900. " " XXXI., 760. Case reported well 8 years.
- ABRAM. 1900. *Journal of Pathology and Bacteriology*, VI., 384. Carcinoma arising in glomeruli.
- ABERCROMBIE. 1880. *Trans. Path. Soc.*, XXXI., 168. Three cases embryonic growth at hilum of both kidneys.
- ADDISON. 1855. "Disease of the Suprarenal Capsules." A collection of the published works of the late Thos. Addison, M.D. Wilks and Daldy. 1868. Vol. XXXVI. of the *New Sydenham Society*.
- AFFLECK AND LEITH. 1896. *Edinburgh Hospital Reports*, IV., 287. Sarcoma of adrenals, references to 20 cases.
- ALBARRAN. 1897. *Ann. des Mal. des Org. Génito-urinaires*, pp. 243, 387. Adenoma and epithelioma.
- ALEXANDER. 1892. *Ziegler's Beiträge*, XI., 145. Structure and function of adrenals, and their relation to the nervous system.
- ALLEN AND CHERRY. Report of Australian Inter. Med. Congress, 1890. Histology of 29 cases of primary renal growth.
- ALM. 1894. B. M. J., ii., epitome 296. Cancer of kidney, hæmaturia 6 years, tumour 3 years, nephrectomy, recovery.
- ALSBERG. 1881. *Langenbeck's Archiv.*, XLIV., 458. Removal of a large lipoma.
- ASKANAZY. 1893. *Ziegler's Beiträge*, XIV., 33. One case of adrenal rest tumour, and remarks.
- BACALOGLY. 1898. *Bull. de la Soc. Anat. de Paris*, XII., 21. Carcinoma, 39 (m.), extensive glandular infection.
- BEADLES. I. 1893. *Trans. Path. Soc.*, XLIV., 98. A large renal tumour found post mortem, hæmaturia.
- II. 1897. " " XLVIII., 241. Adeno-carcinoma found post mortem, with epithelioma of larynx.
- III. 1898. " " XLIX., 179. Infiltrating carcinoma, and four other cases.
- IV. " " " " 260. Fibroadenoma of adrenal.
- V. 1899. " " L., 214. Lesions of adrenals in the insane.

- BENEKE. 1891. Ziegler's Beiträge, IX., 440. One case, Malignant change in adrenal rests.
- BERDACH. 1889. Wien Med. Woch., XXXIX., 357. Diagnosis of adrenal sarcoma, case.
- BERDEZ. 1892. Arch. de Méd. Exper. Paris, IV., 412. Adrenal tumour found post mortem.
- BERGSTRAND. 1897. Ann. des Mal. des Org. Génito-urinaires, XV., 202. Adrenal rest tumour in boy of 8, tumour for a year.
- BIERHOFF. 1900. New York Med. Journal, LXXI., 805. Cystoscopy in a case of carcinoma.
- BIRCH-HIRSCHFELD. I. 1894. Centralblatt f. d. Path. der Harn-Organen. V., 5, 88. Embryonic growths.
II. 1898. Ziegler's Beiträge, XXIV., 343. Histology of embryonic tumours.
- BLACKBURN. 1888. Journal of American Med. Ass., X., 389. Tumour of adrenal found post mortem.
- BORST. 1902. Geschwülstlehre, Wiesbaden. Review and bibliography.
- BOSANQUET. 1901. Trans. Path. Soc., LII., 64. Adrenal cysts found post mortem, references.
- BOYD, STANLEY. 1898. Trans. Path. Soc., XLIX. Malignant papillary adenoma.
- BOYD. Am. Journ. Med. Soc., CXXIII., 1069. Adrenal rest tumour.
- BOWEN. 1897. Journal of Cutaneous and Genito-urinary Diseases, p. 65. Mycosis fungoides.
- BRAULT. 1891. Semaine Médicale, II., 249. Rapid infiltrating growth, great pain, explored.
- BREWER. 1901. Annals of Surgery, XXXIV., 827. Exploration without tumour, well 1 year.
- BRITISH MEDICAL ASSOCIATION. Discussion. B. M. J., 1899, II., 1273.
- BRUCE-CLARKE. Butlin's "Operative Treatment of Malignant Diseases." 1900 edition.
- BRUNS. 1898. Ziegler's Beiträge, XXIV., 375. Spindle-celled sarcoma.
- BUDAY. 1898. Ziegler's Beiträge, XXIV., 501. Occurrence of cysts in adrenal tumours.
- BURKHARDT. 1900. Deutsche Zeitschrift für Chirurgie, LV., 91. Adrenal rest tumours, four cases, review.
- BUSSE. 1899. Virchow's Archiv., CLVII., 346. Fibromyoma. Embryonic tumours.
- BUTLIN. 1887. "Operative Treatment of Malignant Disease." First edition.
- CAILLÉ. 1895. Archives of Pediatrics, XII., 594. Adrenal sarcoma of infant, metastasis in neck.
- CARPENTER. 1902. Rep. Soc. Diseases of Children, II., 69. Adrenal sarcoma in child, æt. 2.
- CHAPMAN. 1897. West London Medical Journal, p. 201. Two cases of renal growth.
- CHAUFFARD. 1896. Bull. et Mém. de la Soc. Méd. des Hôp. de Paris, XIII., 777. Growth of adrenal, multiple fibromata in the skin; pigmentation.
- COHN. 1894. Berlin Klin. Woch., XXXI., 266. Sarcoma of adrenal, girl æt. 9.
- COLEY. 1897. Med. News of Philadelphia, p. 467. Embryonic growth nephrectomy, recurrence in 5 months, Coley's fluid, death in 1 year.

- CRAUFURD, RAYMOND. 1899. *Trans. Path. Soc., L.*, 212. Cyst of adrenal adenoma.
- CURTIS. 1900. *Annals of Surgery*, XXXI., 759. Adrenal growth removed, well 1 year.*
- DALTON. I. 1884. *Trans. Path. Soc., XXXVI.*, 247. Infant, growth of adrenal or liver.
- II. 1899. *King's Hospital Reports*, V., 217. Papillary adenoma, found post mortem.
- DAY. 1880. *Trans. Path. Soc., XXXII.*, 142. Capsular growth $9\frac{1}{4}$ lbs.
- DEAVER. 1902. *Annals of Surgery*, XXXVI., 87. Two nephrectomies for growth.
- DE JONG, JOSSELINE. 1904. *Ziegler's Beiträge*, XXXV., 205. Papilloma.
- DELAFIELD AND PRUDDEN. 1902. *Pathological Anatomy*.
- DELÉTRE AND RAIMONDI. 1897. *Bull. Soc. Anat. Paris*, II., 112. Infiltrating growth, fatal in 2 months.
- DE PAOLI. 1891. *Ziegler's Beiträge*, VIII., 140. Three cases angiosarcoma (? adrenal rest growth).
- DICKINSON v. sub. FOX, COLCOTT. Sarcoma of adrenal in a child, overgrowth of hair, etc.
- DICKINSON, LEM. 1894. *Trans. Path. Soc., XLV.*, 129. Cystic kidney and sarcoma of adrenal in a man æt. 57. Hæmaturia and tumour, 8 years, pigmentation, languor.
- DÖDERLEIN. 1894. *Cbl. f. d. Path. der Harn-Organen*, V., 3. Embryonic growths, statistics.
- DREW. 1897. *Trans. Path. Soc., XLVIII.*, 130. Villous carcinoma of pelvis, papilloma.
- DRIESSEN. 1893. *Ziegler's Beiträge*, XII., 65. Endotheliomata, occurrence of glycogen.
- DUNNING. 1895. *Annals of Surgery*, XXI., 223. Embryonic growth, nephrectomy, recurrence in 9 months.
- EARLE AND WEAVER. 1894. *Journal of Am. Med. Ass.*, page 980. Adrenal sarcoma, æt. 2.
- EASTWOOD. 1902. *Trans. Path. Soc., LIII.*, 153. Adrenal rest tumour in the uterus; review.
- EDMUNDS, 1892. *Trans. Path. Soc., XLIII.*, 89. Cystic adenoma.
- EURICH. 1896. *Journal of Path. and Bact.*, p. 504. Growth composed of adrenal medulla.
- FAGGE, HILTON. 1875. *Trans. Path. Soc., XXVII.*, 204. Report on Case 18 of the Guy's series.
- FAIRBAIRN. 1902. *Trans. Path. Soc., LIII.*, 184. Adrenal rest growth, excision (Bland Sutton, p. 129).
- FENGER. 1898. *Annals of Surgery*, XXX., 386. Nephrectomy for growth, well $7\frac{1}{2}$ years.
- FENWICK, HURRY. I. 1897. *Trans. Med. Soc., XX.*, 237. Papilloma, cystoscopy.
- II. 1903. *Obscure diseases of kidney*. J. and A. Churchill.
- FLETCHER. 1901. *Trans. Path. Soc., LII.*, 199. Embryonic growth.
- FOX, COLCOTT. 1884. *Trans. Path. Soc., XXXVI.*, 460. Sarcoma of adrenals, overgrowth of hair.
- FRÄNKEL. 1886. *Virchow's Archiv.*, CIII., 244. Growth of adrenals, nephritis, uræmia.

- FREITAG. 1898. Ziegler's Beiträge, XXIV., 363. Sarcoma of adult kidney; review.
- FRUITNIGHT. 1895. Archives of Pediatrics, XI., 881. Trial of Coley's fluid.
- GATTI. 1896. Virchow's Archiv., CXLII., 467. Summary of question of adrenal rest tumours.
- GIORDANO. 1892. Ann. des Mal. des Organes Génito-urinaires, X., 584. Growth of pelvis, nephrectomy.
- GRAUPNER. 1898. Ziegler's Beiträge, XXIV., 385. Adrenal rest growth. 399. Kinds of carcinoma.
- GRAWITZ. I. 1883. Virchow's Archiv. XCIII., 39. "On the nature of so-called lipoma."
 II. 1884. Langenbeck's Archiv., XXX., 824. Origin of tumours from adrenal rests.
- GREENHOW. 1866. Trans. Path. Soc., XVIII., 260. Growth of adrenal, no metastases.
- GUILLEMALIN. 1891. Gaz. hebd. de Méd., LII., 612. Methods of diagnosis.
- GUYON. 1900. Ann. des Mal. des Org. Génito-urinaires, XVIII., 1. Diagnosis of renal growth.
- HABERSHON. 1866. Guy's Hospital Reports, XII., 423. Report of Case 15 of Guy's series.
- HANSEMAN. I. 1896. Deutsch. Med. Woch., p. 52. On Endothelioma.
 II. 1902. Zeitschr. f. Klin. Med., XLIV., 1. Tumours, adenoma, endothelioma, etc.
- HAWTHORNE. 1894. Glasgow Med. Journal, XLI., 148. Growth with glandular invasion.
- HEBB. 1895. Westminster Hosp. Reports, IX., 121. Latent growth, sudden embolism.
- HEKTOEN AND RIESMAN'S Text book of Pathology, 1901.
- HERESCO. Thèse de Paris, 1899. Results of 165 nephrectomies for growth, bibliography.
- HEYDER. 1890. Archiv. f. Gynäk., XXXVIII., 301. Growths of the kidney capsule.
- HILDEBRAND. I. 1894. Langenbeck's Archiv., XLVII., 225. Three cases endothelioma, glycogen.
 II. 1894. Langenbeck's Archiv., XLVIII., 343. Various cases.
- HOLMES, BAYARD. 1898. Journal Am. Med. Ass., XXXI., 403. Adrenal rest tumours, review.
- HOLMES. 1872. Trans. Path. Soc., XXIV., 149. Vascular pulsating growth.
- HORN. 1891. Virchow's Archiv., CXXVI., 191. Adrenal rest growths, adenomata, etc.
- LOTT AND WALSHAM. 1893. B. M. J., I., 694. Embryonic growth, removal, recurrence in nine months.
- ISRAEL. I. 1889. Berl. Kl. Woch., CXXV., 156. Methods of palpation.
 II. 1894. Langenbeck's Archiv., XLVII., 302. Twelve nephrectomies for growth, remarks.
 III. 1896. Deutsch. Med. Woch., XXII., 345. Further cases and remarks.
- JACOBSON AND STEWARD. Operations of Surgery. 1902.
- JOHNSON. 1899. Annals of Surgery, XXIX., 329. Three cases of growth.

- JONES. 1891. *Med. Chron.*, Manchester, XXVI. Recurrent papilloma. v. Kelynack, p. 223.
- JONES. 1894. *Deutsch. Med. Woch.*, 209. Two cases of adrenal growth, structure of adrenals.
- KAMMERER. 1891. *New York Med. Journal*, LIII., 79. One case.
1896. *Annals of Surgery*, XXIII., 747. "Cancer" in a woman, æt. 60. tumour 6 years, nephrectomy, accidental death, 1 month, no metastases.
1899. " " XXIX., 753. Nephrectomy, well 5 months.
1901. " " XXXIV., 828. Death from recurrence in 2½ years.
- KEITH. 1902. "Human Embryology and Morphology."
- KELLY. 1898. *Ziegler's Beiträge*, XXIII., 280. Review, three adrenal rest tumours, adenomata, and other cases.
- KEYES. 1903. "Diseases of the genito-urinary organs."
- KELYNACK. "Renal Growths," 1898.
I. 1896. *Journ. Anat. and Phys.*, X., 539. Adrenal adenoma.
II. 1897. *Edin. Med. Journal*, 263. " "
III. 1897. *Med. Chron.*, Manch., 401. Growths of adrenals.
IV. 1897. *Journal Path. and Bact.*, IV., 236. Malignant cyst adenoma (and p. 118).
V. 1902. *Clin. Journal*, XX., 318. Tumours of the kidney.
- KUSTER. I. 1890. *Berl. Kl. Woch.*, XXVII., 641. Death on table from detachment of growth, successful removal of adrenal rest growth.
II. 1897. *Cbl. f. d. Path. der. Harn-Organ.*, VIII., 583. Diagnosis and treatment, exploration.
- LANCEREAUX. 1890. *Union Méd. de Paris*, XLIX., 289. A case.
- LANGHANS. 1890. *Virchow's Archiv.*, CXX., 28. Glycogen contents of tumours.
- LAZARUS. 1894. *Berl. Kl. Woch.*, XXXI., 498. Adrenal sarcoma in a child of 4.
- LAZARUS, BARLOW. 1900. *B. M. J.*, II., 24. Two cases of true unencapsuled lipoma.
- LEVI, EMILY. 1896. *Archives of Pediatrics*, XIII., 97. Table of nephrectomies for embryonic tumour.
- LÖWENHARDT. 1888. *Deutsch. Zeitschr. f. Chir.*, XXVIII., 583. Adrenal rest tumours.
- LUBARSCH. I. 1894. *Virchow's Archiv.*, CXXXV., 149. Adrenal rest tumours, cases, glycogen.
II. 1894. " " CXXXVII., 191. Reply to Sudeck.
III. 1902. *Ztschr. f. Kl. Med.*, XLIV., 491. Reply to Hansemann, characters of adrenal rest tumours.
- LUNN. 1891. *Trans. Path. Soc.*, XLII., 186. Hæmaturia 6 years, no pain, tumour weighed 4½ lbs., no metastases.
- MCBURNIE, 1894, v. sub. Johnson. *Annals of Surgery*, XX., 373. Nephrectomy, boy æt. 10, discussion.

- McCORMAC. 1888. B. M. J., I., 533. Epithelioma and calculus.
- McWEENEY. 1896. B. M. J., I., 323. Two adrenal rest tumours, figures, and review.
- MACKINTOSH. 1895. Lancet, I., 1371. An embryonic case.
- MAIDLOW. 1898. B. M. J., I., 426. Growth removed weighing 7 lbs., nature uncertain, well 3 years. (Bland Sutton, p. 131.)
- MALCOLM. I. 1894. Trans. Clinical Soc., XXVII., 94. Nephrectomy for embryonic tumour, discussion of operation methods.
- II. 1895. " " XXVIII., 287. Child shown well.
- III. 1896. Trans. Path. Soc., XLVII., 119. Sarcoma, girl æt. 19, symptoms 2 years, removal, recurrence.
- IV. 1902. Lancet, II., 1197. Shows embryonic case, well 10 years.
- MANASSE. I. 1895. Virchow's Archiv., CXLII., 164. Adenoma, kinds of carcinoma.
- II. " " " CXLIII., 278. Sarcoma, endothelioma.
- III. 1896. " " " CXLV., 113. Adrenal rest growths.
- MARCHAND. 1883. Virchow's Archiv., XCII., 11. Adrenal rests in broad ligaments.
- MARTIN. 1896. Lyon Méd. One embryonic case.
- MORRIS. I. "Surgical Diseases of the Kidney." 1901
- II. 1893. B. M. J., I., 1. Five nephrectomies.
- III. 1899. " II., 1336. Four cases of adrenal growths.
- MOXON. 1868. Trans. Path. Soc., XX., 385. Adrenal adenoma, size of a chestnut.
- MULLER, HELLMUTH. 1896. Virchow's Archiv., CXLV., 339. Origin of true lipomata.
- MURCHISON. 1870. Trans. Path. Soc., XXI., 240. Bilateral papilloma.
- NEWMAN. 1896. Glasgow Med. Jour., XLV., 179. Carcinoma following stone.
- NORMAN. 1893. Trans. Roy. Acad. Med. Ireland, XI., 377. Tubular adenoma
- OGLE. 1864. Trans. Path. Soc., XVI., 250. Adrenal sarcoma, child æt. 3 overgrowth of hair. (Pitman's case.)
- PARKER, RUSHTON. 1897. Medical Press and Circular, II., 456. Tumour over 7 years, removal, recurrence.
- PAUL. Liverpool Med. Chir. Journal, XIV., 102. Embryonic growths.
- PENROSE. 1893. Trans. Path. Soc., XLIV., 96. Passage of fragments of growth.
- PERRY. 1888. B. M. J., I., 1382. Growth of adrenals.
- PERTHES. 1896. Deutsch. Zeit. f. Chir., XLII., 201. Adrenal rest growths, three nephrectomies.
- PICK. 1901. Berl. Kl. Woch., XXXVIII., 299. On the origin of embryonic growths.
- PICQUÉ. 1902. Bull. de la Soc. de Chirurgie Paris, XXVIII., 895. Successful removal of spindle-celled sarcoma.
- PITMAN. 1865. Lancet, I., 175. Adrenal sarcoma, overgrowth of hair.
- PITT. 1894. Trans. Path. Soc., XLV., 141. Adrenal rests.
- POLLARD. 1884. Trans. Path. Soc., XXXVI., 272. Carcinoma and calculi.
- PONCET. 1897. Lyon Médicale, LXXXV., 122. Transperitoneal nephrectomy.

- RAMSAY, OTTO. 1899. Bull. Johns Hopkins, 20. Review of adrenal growths.
- RICKER. I. 1896. Cbl. f. Allg. Path., VII., 363. Renal elements in adrenal rests, etc.
- II. 1897. „ „ VIII., 417. Adenomata and adrenal rests.
- RINDSKOPF. 1893. Deutsch. Med. Woch., 854. Effect of chloroform on the kidneys.
- RIS, FR. 1891. Beiträge zur. Klin. Chir., VII., 146. Three nephrectomies in adults.
- RITCHIE AND BRUCE. 1890. Trans. Edin. Med. Chir. Soc., IX., 67. Carcinoma of adrenals, secondary in lungs.
- ROBSON, MAYO. I. 1895. Clinical Journal, VII., 98. Two nephrectomies, one in a youth æt. 18.
- II. 1899. B. M. J., II., 1100. Three operations for adrenal growth, review.
- ROBERTSON. 1885. Trans. Glasgow Path. and Clin. Soc., II., 352. Encapsuled nodular growth, general diffusion, illness, death with obscure symptoms.
- ROLLESTON. I. 1895. Goulstonian Lectures on the Adrenals, B. M. J., I. 629.
- „ Trans. Path. Soc., XLVI., 150. Fibro-adenoma of adrenal.
- II. 1899. „ „ L., 214. Cyst of adrenal adenoma.
- ROLLESTON AND KANTHACK. 1894. Journal of Pathology, II., 80. Anomalous mass in kidney.
- ROVSING. 1895. Langenbeck's Archiv., XLIX., 407. Diagnosis and treatment of renal tumours in adults.
- ROYAL COLLEGE OF PHYSICIANS. 1896. Nomenclature of Diseases, p. 335. Classification of tumours.
- RUPPRECHT. 1890. Cbl. fur Gynäk., XIV., 592. Adrenal rest tumour in a child æt. 2½; quoted also in Trans. Path. Soc., XLVII., 128.
- SCHMID. I. 1892. Münch Med. Woch., XXXIX., 256. Embryonic growth, nephrectomy.
- II. 1893. Centralblatt f. Chirurgie, XX., 672. Embryonic nephrectomy, well 3 years.
- SCHMORL. 1891. Ziegler's Beiträge, IX., 523. Adrenal rests in liver and solar plexus.
- SHARKEY. 1881. Trans. Path. Soc., XXXIII., 195. Growth beginning in glomeruli.
- SHATTOCK. 1894. B.M.J., II., 1176. Renal origin of embryonic growths.
- SHORT. 1896. Birmingham Med. Rec., XXXIX., 159. Case, great pain, late tumour, general invasion.
- SLOUKOVENTOFF. 1895. Gazette Méd. de Paris, 581. Growth of adrenal, symptoms pulmonary.
- STEELE. 1896. Med. and Surg. Reporter., LXXIV., 136. Embryonic, well 2 years.
- STEINER. 1896. Berlin Klin. Woch., XXXIII., 565. A nephrectomy by Israel.
- SUDECK. I. 1893. Virchow's Archiv., CXXXIII., 405. Adenoma and carcinoma.
- II. 1894. „ „ CXXXVI., 293. Adrenal rest tumours, reply to Lubarsch.

- SUTTER.** 1902. Virchow's Archiv., CLXVIII., 305. Backward invasion along spermatic vein.
- SUTTON, BLAND.** I. "Tumours, innocent and malignant." 1903.
 II. 1884. Journ. Anat. and Phys., XIX., 458. Adrenal growth in marmot.
 III. 1893. Clinical Journal, III., 4. Analysis of renal tumours.
- TARGETT.** I. 1894. Discussion, Lancet, II., 1096.
 II. 1895. Trans. Path. Soc., XLVII., 122. Adrenal rest tumours of the kidney.
 III. 1897. Trans. Obst. Soc., XXXIX., 157. Adrenal rests in the broad ligaments.
- TERRILLON.** 1890. Bull. et. Mem. Soc. de Chir., XVI., 431
 1890. " " " " XVI., 474
 1891. " " " " XVII., 116
- { Threenodular localized
 growths, weighing
 485, 750, and 1,500
 grs. respectively,
 removed by opera-
 tion. The first case
 was well 28 months
 later.
- THOMA.** 1896. General Pathology, English edition. Black.
- THOMAS, THELWALL.** 1897. Med. Press and Circular, II., 455. Removal of tumour weighing 2 lbs., adrenal rest origin discussed.
- THOMAS, LYNN,** 1904., B. M. J., II., 11. Use of urine separator in a case of growth.
- THORNTON, KNOWSLEY.** I. 1882. Trans. Path. Soc., XXXIV., 141. Successful removal of renal growth.
 II. 1884. " " " " XXXVI., 269. Papilloma and calculus.
 III. 1890. Trans. Clin. Soc., XXIII., 153. Adrenal tumour, woman, æt. 36. Overgrowth of hair, removal, recovery, recurrence in 2 years.
- THURSTON.** 1898. St. Thomas' Hosp. Rep., XXVII., 124. Growth and calculi.
- TREYER.** 1899. Corr. Bl. Schweiz. Aerzte., XXIX., 614. Growth with glandular infection.
- TURNER, CHARLEWOOD.** 1885. Trans. Path. Soc., XXXVI., 464. Growth of adrenals, symptoms pulmonary.
- ULRICH.** 1895. Ziegler's Beiträge, XVIII., 589. Adrenal rests and their growths, lipomata.
- VAN DER BYL.** 1855. Trans. Path. Soc., VII., 268. Embryonic growth.
- VILLENEUVE AND NEPVEN.** 1890. Bull. Soc. Chir. de Paris, XVI., 221. Colloid epithelioma, operation, recovery.
- VOIGT.** 1902. Arch. für Gynäk., LXVI., 658. Malignant papillary adenoma.
- WALKER.** 1897. Annals of Surgery, XXVI., 529. Sarcomata in children.
- WALLACE.** 1902. Trans. Med. Chir. Soc., Edin., XXI., 170. Two cases, an "adenocarcinoma" in a woman, æt. 30, nephrectomy, well in three weeks; and one year later: a sarcoma, man, æt. 53, one year ago hæmaturia, double varicocele, partial excision, varicocele cured.

- WALSHAM, 1885. St. Barth. Hosp. Reps., XXI., 125. Calculus and growth.
- WARTHIN. 1897. Journal of Pathology, IV., 404. Fibrolipoma, 2 lbs., two years' duration, removed.
- WEBER, PARKES. I. 1896. Trans. Path. Soc., XLVII., 117. Lymphosarcoma.
 II. 1898. " " XLIX., 176. Papillary adenoma.
 III. 1899. " " L., 179. Multiple adenomata in contracted kidney,
 " 181. True lipoma.
- WEIR. I. 1887. Annals of Surgery, VI., 38. Papillary cystadenoma, operation, recovery.
 II. 1897. " " XXVI., 128. Calculus, kidney converted into fat.
- WENTWORTH, 1896. Archives of Pediatrics, XIII., 321, 341. Embryonic growths, review.
- WEST. 1879. Trans. Path. Soc., XXX., 419. Growth of adrenal, pulmonary symptoms.
- WHITE, HALE. 1897. Guy's Hospital, Gazette II. Diagnosis of renal growth.
 1884. Trans. Path. Soc., XXXVI., 464. Sarcoma of adrenal, considered secondary.
- WIGLESWORTH. 1892. Liverpool Med. Chir. Journal, XII., 254. Tumour of adrenal, size of an orange, found post mortem.
- WILKS. 1868. Trans. Path. Soc., XX., 224. Fibrous growth of kidney.
- WILLETT. 1895. Trans. Path. Soc., XLVI., 86. (a) Embryonic tumour, recurrence in 11 months. (b) Papillary adenoma successfully removed.

APPENDIX OF CASES.

CASE 1. *Infection of peritoneum, glands, and lungs.* T. G., æt. 18 (m.), was admitted under Dr. Cholmeley, September 8th, 1830, and died September 21st. He had been an invalid several months, with pain, wasting, and a large tumour which reached from hypochondrium to groin on the left side. Post Mortem: there were a few small tubercles (nature undecided) beneath the right pleura, tubercles in both lungs, and in the diaphragmatic pleura. (An examination made in 1890 shows that the main disease here is tuberculous.) The abdomen contained dirty brown fluid and shreds of recent false membrane. The peritoneum was thickly sprinkled with fungoid tubercles of various sizes, like boiled sago. (These were examined in 1893 and found to be spheroidal celled carcinoma.) The descending colon passed over a tumour the size of a half-quartern loaf situated in the kidney. Towards the top a part of this organ was found much distended and stretched, but in other parts it retained its natural structure. Elsewhere nothing could be discovered but the mere tunic distended over the substance of the tumour, which consisted of fungoid material having the consistency of mortar, yellow, and in some few points semi-transparent. Traces of cysts and debris of vessels were to be distinguished in it. Pelvis healthy. Other kidney healthy. The absorbent glands near it, and those about the aorta, were loaded with opaque fungoid material and fungoid tubercles. Liver and spleen normal, their peritoneal covering tuberculated, as the peritoneum elsewhere. *Green book X. 47.*

CASE 2. *Short history, glands invaded.*—A. K. M., æt. 67, was admitted under Mr. Cooper, July 30th, 1834, and died August 14th. He had a fall four months ago, injured his loins, and coughed up some blood. A few days ago he had retention; the catheter brought away chocolate-coloured bloody urine. Hæmaturia continued, and was accompanied by pain in the back and left side. He died with a bloody diarrhœa. Post Mortem: Lungs emphysematous and congested. On one of the bronchi of the left lung a mass, the size of a walnut, whitish, breaking down in places. Liver—one small solid deposit on the surface. Right kidney large, ureter dilated and thin, “a little gut.” Left kidney enlarged, adjoining it a mass like a kidney in size and shape surrounding the aorta. The upper half of the kidney contained a scirrhus mass which rendered it twice its normal size. This seemed to have developed in the kidney, and to have encroached on its natural structure, (destroying a full half), from which it was ill-defined. It was rounded and nodular in form, a soft scirrhus growth with cyst formation and parts of almost medullary softening. There was some appearance of a ragged vegetation sprouting into the superior infundibula. The mass round the aorta consisted originally of glands, one was breaking down into a large cyst. *Green book XVII. 140.*

CASE 3. *Growth of adrenal, two months' pain, invasion of liver, lungs, and glands.*—S. B., æt. 29 (m.), was admitted under Dr. Addison on November 2nd, 1842, and died on December 20th. Two months ago he had pain in the left loin and could not move his leg freely—no other symptoms. A tumour was felt on admission at the lower margin of the liver, which increased in size daily. He referred to the loin as the seat of pain. November 15th, explored by trocar—a few drops of blood issued—died five days later. Post Mortem: a few breaking-down masses in the lungs the size of marbles. Liver a mass of growth. Mesenteric glands enlarged. A firm oval mass consisting of fungoid disease of the suprarenal body, cerebriform, but having oval portions the size of nuts, of cheesy consistency. Kidneys healthy, the whole reached to and thrust up the spleen. *Miscellaneous Inspections XXXII.* 83.

CASE 4. *Embryonic growth, invading vena cava and auricle.*—M. W., æt. 2 years 9 months (f.) Admitted under Dr. Addison, January 25th, 1845, and died January 26th. Scarlatina 9 months ago, blow on side 6 months ago, history of vomiting. Post Mortem: Lungs free. Right kidney bigger than two adult kidneys. A good part of its tissue detached by 2·3 ozs. of very bloody fluid. The kidney retained a good deal of its form and tissue, being expanded by the growth within, which had escaped widely downwards as a hæmatoid mass, the rest medullary. "Vena cava and both renal veins dilated with very soft and bloody fungus, adherent, distinctly a growth, i.e., organized into the right hepatic veins and auricle, a round nob the size of a walnut almost a clot of black blood, but containing serous cells or cysts, the whole variously cerebriform, fluid, and bloody." Left kidney, soft, pale, and dull. Liver, a probable deposit posteriorly. Spleen, "ill defined peas—perhaps cerebriform." *Miscellaneous Inspections XXIII.* 91.

CASE 5. *Growth spreading by vena cava to pulmonary artery.*—W. M., æt. 40 (m.). Admitted under Dr. Addison, March 3rd, and died October 10th. He had been "ill" for 12 months; urine free from hæmatozine. Post Mortem: Right lung singularly full of water, and all the first branches of the pulmonary artery filled with pale brain-like fungus. The pericardium contained red fluid and scabrous fibrin. Left kidney nearly half the size of a liver, very full of fungus, cerebriform, or like soft cartilage. Pelvis filled with fungus. Calyces 2 inches long. Left renal vein and an inch of vena cava full of fungus, and above two or three fingers of organized growth—pale, fragile, and unequal. Right kidney enlarged, free. Liver contained one dark red tuber the size of a bean. *Miscellaneous Inspections XXXIII.* 205.

CASE 6. *Growth, calculi, direct invasion extending through diaphragm, abnormal adrenal.*—J. S., æt. 35 (m.). Admitted under Dr. Gull, December 1st, 1852, and died January 13th, 1853. No history is available. Post Mortem: On opening the peritoneum a large fungoid mass was seen passing up from beneath the liver, but unconnected with it, passing up under the stomach and invading its outer coats but not its mucous membrane. It arose from the left kidney, the whole of which was infiltrated with growth. It invaded the intercostal muscles and ribs 11 and 12, and extended backwards through the muscles, forming an external tumour. The kidney presented a mass of growth, dead in the centre, sprouting at its circumference. The pelvis contained an

irregular nodulated calculus of pelvic shape; four others occupied cysts or dilated calyces beneath the capsule. The capsule was not invaded except at the hilum. The malignant mass passed through the diaphragm, invaded the left pleura (causing pleurisy), and in one or two places reached the lung. Right lung, one or two small deposits on the surface. Liver, 12 separate deposits. Right kidney healthy, $1\frac{1}{2}$ times the normal size. The suprarenal capsule was adherent to the body of the kidney and apparently beneath the tissue, but at the position where it was adherent the cortex appeared contracted; whether this was the result of inflammation or an abnormal position it is difficult to say. *Miscellaneous Inspections XXVII. 35.*

CASE 7. *Two years' history of tumour, metastases in lungs and bones, glands free.*—T. H., æt. 69 (m.). Admitted under Dr. Addison, February 18th, 1857, and died April 23rd. Well until two years ago, when he felt tightness in his abdomen, a tumour discovered after some months' ill-health. On admission very feeble. Large mass, evidently renal, in right side of abdomen, which can be moved bimanually. Hæmaturia. Post Mortem: Lungs and pleura full of growth, bronchial glands free. The tumour is almost all kidney, very few of the adjacent glands are involved. The ascending colon passes in front of it, the vena cava and aorta are displaced to the left. Mesenteric glands free. One mass the size of an egg in the right lobe of the liver adjoining the kidney. Left adrenal large, one deposit the size of a pea. Right adrenal involved in the growth, and forming a lump the size of an egg on top of it (the growth had evidently begun in both adrenals from a localised deposit). At one end remains of the capsule are seen, about one-half was destroyed. Right kidney quite circumscribed, and can be turned out easily with the adrenal and a few infected lymphatic glands, the whole the size of a fetal head. On section a firm and fairly uniform mass of growth, some soft and growing, some breaking down and caseating. On closer examination a bit of healthy kidney was still seen intact, and in parts the mucous membrane of the pelvis remained, so that when the growth was removed the original form of the organ could be seen, and the method of invasion. Ureter normal and free, its channel joined the cancer in the pelvis. Renal vein full of a mass of soft growth, protruding into the vena cava. Nodule in right epididymis. Deposit in inner condyle of right humerus, destroying the joint. Growth from medulla of right tibia. *Insp.* 1857, 80.

CASE 8. *Five months' pressure on nerves, no metastases.*—M. R., æt. 57 (m.). Admitted under Dr. Habershon, July 16th, 1856, and died October 29th. gouty—a drinker. In May last had sudden pain in course of left dorsal nerve, apparently causeless, which grew worse. He was admitted to hospital, no disease could be found, urine normal—discharged unrelieved. Re-admitted under Drs. Habershon and Hughes, September 13th. Relieved by blisters. October 22nd. became soporous, urine drawn off was scanty and highly albuminous, died a week later. Post Mortem: Lungs normal; heart, some vegetations on mitral and aortic valves. Mesentery and glands normal, liver cirrhotic. Peritoneal surface of spleen covered by whitish tubercular layer $\frac{1}{2}$ -in. thick, cancerous (? capsulitis). In the vena cava a loose ante-mortem clot an inch long, opposite renal veins, softened centrally. Right kidney engorged, patch on surface such as is seen with a diseased mitral—nothing cancerous. Left

kidney, substance engorged, tubules filled with granules and cells, surface granular. Pelvis nearly obliterated by cancerous growths. Thickening round pelvis leading to pressure on left dorsal nerve. At the pelvis were several masses the size of walnuts, two had dense fibrous envelopes and cheesy degenerative contents of fat granules and nuclei. Adjoining and encroaching on the kidney was a rather larger mass, softened from effused blood, and presenting under the microscope abundant large nuclei and blood. In the kidney structure near this part was a small mass of effused yellowish-white product, consisting of large cells, some containing two large nuclei, others large nuclei nearly filling the cells, others free. *Insp.* 1856, 210.

CASE 9. *Hæmaturia 3m., paralysis 2m., infection of glands, spine, and lungs.*—S. W., æt. 58 (f.). Admitted under Dr. Hughes, December 5th, 1857, and died January 21st, 1858. She was admitted for hæmaturia of three months' duration. She had been ailing for about a year, and for two months had been unable to walk. On admission paraplegia increased and bed-sores appeared. Post Mortem: Body much wasted. The cancerous growth in the abdomen had destroyed a large part of the spine. It seemed as if the bodies had been attacked separately, as two of them had quite disappeared and the intervertebral discs were in contact. Above this the bones were black and necrosed, and connected with some suppuration in the back. The lower dorsal and upper lumbar vertebræ were thus diseased, and the disease had extended into the spinal canal. The membranes of the cord were black and sloughy, and the medulla within was quite soft. Both lungs contained numerous nodules of cancer, mostly on the surface, one or two pedunculated; bronchial glands infected. Liver, peritoneum, pancreas, normal. The lumbar glands formed a large malignant mass. *Museum specimen* 1649.

The left kidney is somewhat enlarged and laid open to show its substance thickly beset with soft nodules of growth $\frac{3}{8}$ -1 in. across. On the reverse, similar nodules are seen to project from the surface of the organ, and there is one prominent bossy growth measuring $2\frac{1}{2}$ in. across, to which the capsule is adherent. Histologically the deposit consists of a fibrous stroma, the alveoli of which are lined with cubical epithelium, some of them being also filled with spheroidal cells. "Cylindrical and spheroidal carcinoma." *Insp* 1857, 11.

CASE 10. *A year's vague illness—extensive glandular infection—later invasion of lungs.*—W. G., æt. 30 (m.). Admitted under Dr. Willis, January 11th, and died February 1st. He had been ailing for about a year, and gradually becoming anæmic and weak, but with no positive symptoms. He had been considered phthisical, but the physical signs of this were wanting. On admission he was bedridden and anæmic, but had no pain or symptoms to localise his illness; there was some inconsiderable crepitus at the apices of his lungs. Idiopathic anæmia, Addison's disease, and deep-rooted disease about the stomach were discussed. Post Mortem: Wasted; right leg swollen. Small masses of recent growth scattered throughout the lungs. At the apices old healed phthisical cavities, and a few minute tubercles. Bronchial glands full of caseous deposits. Liver normal. Right kidney much enlarged and nearly destroyed by growth, which was probably primary here. The growth passed to the hilus but did not break through it, and it was still outside the

vein, though the latter was compressed. Ureter and pelvis unaffected. The neighbouring glands and the whole lumbar chain were infected, and through these the deep inguinal below, and the mediastinal above. Vena cava compressed and nearly blocked by adherent thrombus; there was no growth within the vein. Glands round pancreas very firm; no doubt their pressure on the thoracic duct had been a cause of emaciation. Small deposits in left adrenal; left kidney normal. *Insp.* 24.

CASE 11. *Vague history—metastases in lungs—no others.*—E. P., æt. 62 (f.). Admitted under Dr. Barlow, March 5th, 1861, and died May 17th. She came in with a history of pain in the chest and diarrhœa, and said that some time ago she used to throw up everything she took. She was wasted. Albuminuria was present and œdema of legs. She continued to have diarrhœa and occasional vomiting. (Report ends.) Post Mortem: Body wasted. Numerous small secondary deposits in both lungs. Liver, glands, adrenals, normal. Other kidney not described. Right kidney much enlarged by growth, the upper half replaced by a round tumour, which had not come through the capsule, but within projected into the pelvis. On section soft and medullary, the interior containing yellow degenerating masses. *Insp.* 1861, 92.

CASE 12. *Embryonic tumour, no metastases.*—T. H., æt. 11 (m.). Admitted under Dr. Gull, February 11th, 1863; died February 27th. He was sent up from Swindon with a large abdominal tumour, which had been growing for some months. It sprang from the left side, and presented obscure fluctuation. The boy was ill and thin. Mr. Birkett explored and extracted a little gelatinous matter. Post Mortem: Body very thin. Lungs normal. Large tumour present in left loin, reaching from diaphragm to pelvis, and nearly reaching the right side. It touched the parietes in front, so that the intestines lay coiled up in a small space on the right side and the transverse colon above. The descending colon ran down the front aspect of the tumour. On examining the posterior surface, the left kidney was seen stretched over the tumour, and about twice its usual size and length; its upper part was free, its lower part involved in the disease. The anterior part of the organ was almost destroyed, especially below; above the upper part was untouched, even its anterior margin. Contents semi-fluid. The growth at the lower part seemed at first sight to be soft cancer, but emitted no juice on pressure, and corresponded rather to recurrent fibroid. Some parts had a gelatinous fluid in the meshes. *Insp.* 1863, 51.

CASE 13. *Tumour 2½ m. hydronephrosis, glandular and slight lung invasion.*—W. S., æt. 60 (m.). Admitted under Dr. Barlow May 26th 1864, died August 21st. Six weeks ago he noticed a swelling the size of the palm of the hand below his left ribs, fixed, not moving with respiration. Urine acid, no albumen. Tumour increased and became painful, and evidently contained fluid. On July 24th there was pus in the urine. Mr. Foster put in a trocar, and drew off 2-3 pints of bloody fluid. The cyst soon filled again, and the patient sank and died. Post Mortem: A few deposits in lungs. The tumour was clearly an enlarged carcinomatous kidney. The great bulk of it was a cyst chiefly filled with blood. When this was emptied, white masses of fibrin were seen adherent to its walls, and nodules of growth in them. The lower

part of the kidney was composed of fungous masses, and at the upper end, where remains of kidney substance were seen, some distinct masses of cancer existed. These were connected with similar masses in the lumbar glands. *Med. Reports p. 34. Insp. 1864, 207.*

CASE 14. *Paralysis, deposits in skull, glands, liver.*—J. R., æt. 34 (m.). Admitted under Mr. Cock, October 21st, 1863, and died November 11th. He was admitted with partial paralysis, attributed to an injury to his back, and lived three weeks. His mind was enfeebled. Post Mortem: Numerous cancerous deposits between skull and dura mater. Spine not examined. Numerous patches of tubercle all over the surface of both lungs. Cancerous deposits in glands near stomach. Liver full of deposit, cirrhotic, and hard. The deposits in lumbar glands, kidney, etc., involved the adrenals. These were 2-3 times their usual size, and at first sight seemed quite destroyed. A section showed a mass of effused blood, with white growth round the edges, and covered in one case partially, the other completely, by flattened cortex. On the top of one kidney and in contact with the suprarenal body was a large tumour, bigger than the kidney itself, containing a quantity of debris, and in a few places well-marked growth. A careful section showed that the cancer had grown in the kidney, destroyed its upper part, and expanded the true capsule of the kidney over it. *Insp. 1863, 271.*

CASE 15. *Growth of adrenals. Weakness, vomiting, pain, local invasion, deposits in both kidneys and ovaries.*—E. B., æt. 38 (f.). Admitted under Dr. Habershon, June 16th, 1866. Her health had been failing for 8 years, and the duration of the growth was uncertain. The most marked symptoms were abdominal pain, emaciation, loss of appetite, weakness, vomiting, sallowness, and the presence of a tumour. Post Mortem: Lungs normal. No evidence of pressure on thoracic duct. Two large tumours connected across spine by enlarged glands and pushing cola forwards, extending above kidneys, and pushing up liver and spleen. Liver flattened and capsule invaded over an area 5 inches in diameter, where it touched the mass. Spleen not adherent. Both adrenals enlarged to 3-4 times the bulk of a kidney. Both kidneys separable, the right touching at one point only, the left applying itself to a convexity of the lower end of its adrenal. Kidneys irregular, almost destroyed by masses of growth developing in the cortex. Only the lower part of the right kidney normal. Growth encephaloid. Liver free. Glands near the growth enlarged. Both ovaries, the size of a walnut and kidney potato respectively, were infected with growth. *Insp. 1866, 177. Guy's Hosp. Rep. XII., 423.*

CASE 16. *Death from metastases in brain and thorax, abdominal glands free.*—G. C., æt. 51 (m). Admitted under Dr. Habershon, February 22nd 1870, and died March 10th, 1870. Seven months ago he fell and hurt his back, lately he has had loss of memory and fits in which he does not lose consciousness. Paralysis of right side followed; he became nearly comatose, but could protrude his tongue when asked. He lost his speech, either from aphasia or dementia. Urine never procured. Post Mortem: Left side of brain much flattened. A spherical tumour occupied the left vertex, the size of a billiard ball, containing cysts, vascular, with much cheesy degeneration. Round

it the hemisphere for two-thirds of its extent was in a state of yellow softening. In the pleura some 20 growths, the size of a pea to a bean. Lungs themselves free. Bronchial glands infected. Vena cava stuffed like a small sausage with ante mortem clot of various ages. Evidently there had till recently been a way for blood to creep through the interstices of the clot, which extended down to the iliac veins. Liver, mesenteric, and lumbar glands quite free. Adrenals free. Right kidney, 30 oz. swollen into a cancerous mass, which was still within the capsule at nearly all points, but at some few was growing through as fungoid nodules. On section a good deal of necrosis, cysts few and small. Veins of kidney full of ante mortem clots, and the pelvis filled by a dark brown decolorizing clot which extended down to the upper end of the ureter. Here the cancer sprouted through into the tube and obstructed it, causing distension of the pelvis. The cancer in the ureter had a soft papillary growth, its growing margin in the kidney was in the form of soft red vascular masses, it came chiefly around the roots of the pyramids as marble-sized spherical growths. Testes normal. *Insp.* 1870, 54.

CASE 17. *Hæmaturia 21 months, tumour 1 month—invasion of liver and some glands.*—J. C., æt. 34 (m.). Admitted under Dr. Habershon May 6th 1879, and died May 27th. Twenty months ago he had hæmaturia, and since then he has lost much flesh. A month ago he first noticed a tightness of the abdomen after meals, this got worse and the hepatic region became tender. On May 1st he again had hæmaturia. On admission there was a tumour occupying the right hypochondriac, lumbar, and iliac regions. The epigastrium was dull. Urine dark, 1028, small red clots. While in the hospital he had an interval free from hæmaturia, and then a recurrence. Tenderness and swelling increased. On the 26th while walking across the ward he fainted and died in a few minutes. Post Mortem: Pleuræ healthy, and lungs, except for some compression of the right base. Heart normal. Liver, 148 oz., full of large masses of growth, some circumscribed, some infiltrating, all of them diffuent and creamy. Lumbar glands a little large and very cream-like on section. Adrenals normal. Left kidney, 11 oz., dark and hard enough for the kidney of cardiac obstruction. The vessels are healthy and not blocked by the tumour. Right kidney, 69 oz., filling right loin and crossing the mid-line, ascending colon pushed forward and adherent to the front of it. Right lobe of liver adherent and displaced upwards. Surface lobulated, growth encapsuled except where it adheres to the liver. Vessels not plugged or abnormal. On section a large fibrocystic mass retaining the shape of the kidney, but with no trace of renal tissue. Cysts full of soft red and brown creamy fluid. Right hydrocele. Testes normal. Microscopically: cells of all shapes, many angular or epithelioid. "Therefore the growth may be called medullary carcinoma." *Insp.* 1879, 202.

CASE 18. *Bronchitis—growth found post mortem—no metastases.*—M. H., æt. 67 (f.). Admitted under Dr. Pavy, July 9th, 1873, and died July 14th, 1873. She had had a cough 18 years, and for the last month, œdema of the legs. On admission she was thin, with the signs of bronchitis. Œdema of legs and of the body wall, chiefly on left side; ascites. Liver palpable below ribs, Urine, 1022, albumen, granular casts. Post Mortem: pleurisy, emphysema, œdema of lungs, bronchiectasis, a dissecting varix of left femoral vein. Liver,

vena cava, etc., normal. Left kidney, $4\frac{1}{2}$ ozs. granular, some small cysts, surface speckled with minute yellow grains, which microscopically were of some solid opaque fatty material. Right kidney, 7 ozs., smooth, with similar yellow points, Described in the catalogue as "a kidney of normal size, of which the structure is for the most part replaced by a mass of growth, which in the recent state was soft and yellowish. On section the growth consists of an aggregation of nodules partially separated from each other by fibrous tissue. The pelvis is dilated, and at either end of the kidney is a shell of persistent cortical substance. On the reverse a mass of growth is seen occupying the hilum and projecting into the renal vein." It was noted at the post mortem that in addition to this another mass the size of a walnut lay in the channel of another dilated bunch of veins, probably going to join the trunk outside the organ. Even within the hilum it was evident that a good deal of the mass lay within dilated venous channels. Histologically the growth is a medullary spheroidal-celled carcinoma with areas of colloid degeneration. In the post mortem report the growth is described as full of fat globules, looking like a very fatty liver. After a time however, it became evident that these globules were inside cells of a very irregular form, with large oval nuclei. Some were of the shape of cylindrical epithelium, some pear-shaped, some granulated. The growth was not suppurating but infiltrated with fat. Even the most recently growing parts presented the same peculiarity. In the report to the Pathological Society it is noted that the cells were not degenerated but infiltrated with fat. On hardening, a portion shewed the characteristic alveolar structure of a carcinoma. Many cells contained two nuclei. *Insp.* 1873, 219. *Museum Specimen* 1647. *Med. Rep.* 194. *Trans. Path. Soc.* XXVII., 204.

CASE 19. *Two years hæmaturia—invasion of portal venous system.*—W. H., æt. 47 (m.). Admitted under Dr. Willis, January 4th, and died February 20th. He has had hæmaturia two years, and has been admitted three times previously. For the last 9 months he has been able to walk ten miles daily. He now comes in for hæmaturia, ascites, and pain at the umbilicus and bladder. The abdomen is distended and dull, except over the stomach and descending colon. A mass fills the left lumbar and part of the left iliac and hypochondriac regions. Abdominal veins distended. Edge of spleen palpable over the tumour. Urine 1026—turbid—no albumen. Only about half-a-pint is passed daily. Ascites and pain increased, nausea came on, and œdema of the lower half of the body. He gradually sank and died. Post Mortem: There was one infected gland behind the manubrium, and a deposit the size of a marble in the left lung. Otherwise the thorax was healthy. The vena cava inferior contained clot of 3-4 weeks' duration, reaching down the iliac and femoral veins. Peritoneum free, it "was projected highly in spots, but did not take on the cancer action." The mesenteric veins were distended with cancer down to the branches which ran over the intestinal walls; they were thickened and contained a pink creamy diffuent material. The veins were involved where their branches crossed the cancerous kidney: here the cancer thrust itself up in large masses, which were softened within to a cyst-like appearance. The liver was full of cancer. . . . It appeared certain that the cancer came generally into the liver along the portal channel. In the same way it reached the colon also, and from the ends of the cancerous veins it grew into the mucous membrane of the bowel in heap-like elevations showing a remarkable

preference for the mucous rather than the peritoneal surface. Left adrenal involved in the growth. The left kidney formed a mass the size of a cocoon, the colon passed down in front of it and was adherent at one point. The organ preserved its general outline and had a thick capsule. There were effusions of blood into the remains of the pelvis and on the surface of the section. The left testis contained a nodule of growth. *Insp.* 1874, 51. *Med. Rep.* 342.

CASE 20. *Hæmaturia 9 months, hydronephrosis—local invasion without metastases.*—J. C., æt. 66 (m.). Admitted May 12th, 1873; re-admitted under Dr. Moxon December 17th 1873, and died February 27th, 1874. He has had bronchitis five years. Twenty years ago he had a sudden attack of hæmaturia. In May, 1873, he passed blood. He was twice admitted to hospital for this. In June he noticed pain, and there was a dull elastic fulness in the left hypochondrium. Bleeding then ceased, but recurred in August, and again later. On re-admission it had been copious for the last seven weeks. There was then pain in the left side, and a dull rounded mass in the left loin. Liver dulness normal. Systolic aortic murmur. Urine was 30-40 oz. daily, alkaline, 1020. It contained blood, but no clots or casts. Bleeding and pain continued. During January the daily urine sank to 14 ozs. and became purulent. The tumour became rather bigger and more distinct. Post Mortem: Pleura and lungs free. Aortic stenosis. Mitral failure. Fibroid degeneration of left ventricle. Liver free, perihepatitis. No glands or thrombosis mentioned. Capsulitis of spleen. The left kidney formed a flattened tumour over which ran the descending colon. It contained two collections of reddish purulent urine. On emptying these the lower part collapsed; the upper remained as a solid mass of firm whitish growth infiltrating the structures to the inner side of the kidney and the side of the spine and round the aorta, which was at one place completely surrounded. The lower part was an ordinary sacculated kidney, without secreting structures. The new growth was in places growing into the septa, and even fungating into the cavity. The growth was doubtless in the kidney; its exact site could not be made out. It infiltrated the surrounding structures, and the glands round the aorta were full of it. Elsewhere no secondary growths. Right kidney granular, arteriosclerosis. *Insp.* 1874, 76. *Med. Rep.* 300.

CASE 21. *Growth found post mortem, death from fractured base.*—J. C., æt. 55 (m.). Admitted and died December 13th, 1875, under Mr. Bryant. He fell, fractured the base of his skull, and died. The post mortem showed the condition diagnosed. Apart from this, the left kidney was occupied at its upper and hinder part by a lobulated cystic tumour as big as an orange. It proved to be solid and composed of a gelatinous alveolated tissue with blood extravasated into its cortex. It was distinctly in the substance of the organ. It was very soft, and seemed hardly organised at all. In the pyramids of the kidney were many cysts filled with the same mucous material. Fat at the hilus free. The rest of the kidney and the adrenals were healthy. Microscopically the growth is cellular and alveolar, the cells are very difficult to recognise owing to the peculiar glassy change they have undergone. It is described as colloid and cylindrical celled carcinoma. *Insp.* 1875, 494. *Museum Specimen*, 1650.

CASE 22. *Growth of adrenal, local and glandular infection, extension through diaphragm.*—J. S., æt. 25 (m.). Admitted under Dr. Pavy, April 19th, 1876, and died June 7th. Perfectly well up to six weeks ago. He then, while digging, was seized with a sudden pain in the left side, which forced him to lie down, and since then he has been in bed with continuous pain, aggravated by solid food, and some vomiting. On admission he was sallow and wasted, his abdomen was not distended, but there was a painful hard solid mass, resonant on percussion, in the left hypochondrium. Urine normal. Heart and lungs normal. Pain went on, one day he had retention, which passed off. The wasting increased, and the tumour became rather less definite in outline. On April 24th he developed a left-sided pleurisy. On June 5th his liver was noticed to be hard and enlarged. Just before death he passed a quantity of blood from the bowel. Post Mortem: Diaphragmatic pleurisy right side (slight). Left pleura contained two or three pints of fluid. On the surface of the diaphragm was a layer of fleshy growth, which had extended through it. Lungs free, the left compressed. Mediastinal glands all enlarged and fleshy. The left side of the diaphragm infiltrated by growth. Kidneys normal. A large mass was present in the left hypochondrium, pushing forward the stomach and transverse colon. On separating the coils of intestine below this a cavity was opened full of dirty fæcal fluid. An acute peritonitis was present, possibly due to escape from this, the surfaces were covered with lymph, and there was half-a-pint of fluid in the pelvis. Mesenteric and lumbar glands enlarged, white, and succulent. The spleen had to be shelled out of the mass, and the upper end of the kidney was embedded in it. It extended beyond the stomach, which adhered to it, involved parts of the pancreas, and merged into a mass of enlarged glands which lay along the front of the spine. It surrounded, but did not invade, the portal vessels. The adrenal was much enlarged and converted into a fleshy mass of growth. It seemed probable the disease had begun in this organ. Right adrenal normal. No communication could be found between growth and intestine, and the terminal hæmorrhage is unaccounted for. *Insp.* 1876, 223. *Med. Rep.* 270.

CASE 23. *Hæmaturia over 2 years. No secondary invasion.*—H. D., æt. 69 (m.). Admitted under Dr. Habershon, March 13th, 1876; re-admitted May 16th, 1878, and died May 18th. Since 1873 occasional nausea and malaise. Early in March 1876 he suddenly felt sick and passed half-a-pint of bright blood, and later blood mixed with urine. He developed a cough and a shooting pain in the left loin. Bleeding was increased by exertion. At his first admission there was tenderness in the left loin and hæmaturia. He remained in nearly three months, and on one occasion it was noted that he passed some villous growth enclosed in a clot. After his discharge he had hæmaturia with intermissions of as long as six weeks. On May 16th, 1878, he had suppression of urine for 36 hours. He was admitted, and three pints of blood were drawn off with a catheter at different times during the next two days. Post Mortem: Lungs, liver, lumbar glands, adrenals, normal. Right kidney fully twice the normal size, nodulated on the surface, and two-thirds of it occupied by a soft yellow alveolated growth. It seems to have started in the cortex, for the pyramids are still discernible in the growths almost untouched. There is part of the lower end of the kidney which is uninvaded by growth—this is granular

on the surface. The other kidney is granular and both contain cysts. The arteries are thick. Prostate the size of a Tangerine orange. *Insp.* 1878, 187. *Med. Rep.* 338.

CASE 24. *Hæmaturia 3 years, metastases in bones.*—W. A., æt. 34 (m.). Admitted under Dr. Moxon, June 15th, 1874; died August 4th. He has hardly been sober for five years, except when he was ill in bed. Quite well till three years ago, when he had hæmaturia. He has had two attacks since, one 2½ years and one 2 months ago. A month ago he woke up with "pins and needles" and total paralysis of left arm and shoulder, present since. On admission cannot move left arm. Pain in left fore-quarter. Weakness left knee. Pain in head. Chest normal. Urine 1012 no albamens. The left arm improved a little in hospital, then got worse, and the right arm became weak also. A "postpharyngeal abscess" of 4 oz. was evacuated. His legs became weak and entirely paralysed by the end of July. Two "cold abscesses" appeared on the left side of his chest. Post Mortem: Many deposits of growth in both vault and base of cranium, and one in the 4-5 cervical vertebræ, infiltrating the tissues in front. Two ribs on the right side had masses replacing their centre. Pleura and lungs normal. On the anterior surface of the right auricle a growth the size of a pea, 3-4 nodules in the liver. A few lumbar glands adjoining the left kidney were slightly enlarged with white growth. A secondary nodule in each adrenal. Right kidney normal. Left kidney a large mass of soft cancer, triangular in shape, the lower end globular. The mass was composed of red, white, and yellow substance in various stages of degeneration. At the upper end some kidney substance was spread out over the growth, as if it had began near the pelvis and made its way outward. The pelvis was much dilated, and large polypoid masses of growth projected into it. The renal vein was invaded and blocked by a mass of growth. Microscopically the growth was composed of all kinds of cells, mostly angular and epithelioid, of all sizes. The stroma was fibrous, and did not tend to a definite areolation. *Insp.* 1874, 317. *Med. Rep.* 92.

CASE 25. *Tumour 12 years, hæmaturia 1½ years—General visceral infection.*—H. S. æt., 49 (m.). Admitted under Dr. Moxon, August 2nd, 1876, and died September 4th, 1876. Twelve years ago he noticed a small movable lump the size of a pigeon's egg in the left lumbar region, which was painless. Fifteen months ago he had intermittent hæmaturia over some 3 weeks, this has not recurred since. Seven months ago he began to lose flesh, and 6 weeks ago he noticed a rapid increase in the size of the lump, and an aching in the legs. He thinks this is the same lump that he noticed originally, not a fresh one. For the last week it has been painful. On admission he was wasted, the left side of the abdomen from ribs to ilium was filled with a solid dull mass, crossed vertically by the descending colon, nodular, and movable on bimanual palpation. Urine, 1015, albumen, no blood. He became weaker, suffered from a varying amount of pain, and on August 14th, became delirious and remained so till his death. The tumour increased in size. Post Mortem: lungs full of secondary nodules, soft, most of them under ½-inch across. Liver, 62 oz. a few scattered nodules. Mesenteric glands much enlarged with deposit, some of which is caseating. Left kidney 47 oz., a mass of cancer arranged to some extent in nodular masses. A large part of this had undergone caseation, and there were also fibrous patches. The recent growth was soft and pulpy.

Some cysts contained a dark brown fluid. No renal substance left. Right kidney, several little nodules of secondary deposit. *Insp.* 1876, 363. *Med. Rep.* 323.

CASE 26. *Rapid glandular invasion—death in three months.*—M. L. (f.), æt. 41. Admitted under Dr. Willis, September 11th, and died September 27th. Family and personal history good. Thirteen children besides miscarriages, Only three alive, of whom two are hydrocephalic idiots and one deranged. The others died of hydrocephalus at about two or three years of age. On June 30th. she had a severe labour. She got up three weeks later, and was attacked with shivering, vomiting, and pain in right loin and epigastrium. She went to bed for a week and then noticed a swelling in the right side. On admission she was wasted, the skin dry and pigmented, ankles swollen, nausea constant. Urine 1030, acid, albuminous. Right side of abdomen hyperæsthetic. Hard tender mass continuous with liver. This is dull and the rest of the abdomen tympanitic. She lay in a listless state with occasional vomiting (no jaundice), and died with signs of heart failure. Post Mortem: the whole dorsal and lumbar spine was infiltrated with growth, which also involved the right lower ribs and subpleural tissue. Both lungs were studded with nodules of hard growth, in size from a pea to a walnut, which were more numerous on the surface. Blood-stained fluid in right pleura. Bronchial glands unaffected. Liver enlarged and studded with soft growth, especially on the right side. The liver, right kidney, glands, aorta, and vena cava were fused into one shapeless mass of growth. The vessels were compressed but not involved, and a clot which filled the left common iliac vein was apparently innocent. There was a nodule in the ilium near the ilio-cæcal valve. Right kidney showed a fairly healthy structure below and inside, where it was wasted and anæmic. The growth was soft and medullary, and looked oldest above. There was one small nodule in the left kidney, and of the adrenals the right was cancerous, the left healthy. Dr. Willis thought the right kidney the primary source, no other possible origin was found. *Insp.* 1877, 361. *Med. Rep.* 309.

CASE 27. *Calculus 4 years, hæmaturia, direct extension to liver, growth in ureter, no other metastases.*—W. P., æt. 58 (m.). Admitted under Dr. Moxon, May 1st, 1878, and died July 11th, 1878. Four years ago he had hæmaturia and pain. Three years ago he passed a calculus the size of a pea; he continued to pass blood up to the present. In February he was seized suddenly with severe pain in the right side while in bed. In April he had dysuria, the passage of clots, and pain, which has continued. There is no vomiting, appetite is good, the liver normal; urine 1022, contains blood. A mass is felt bimanually deep in right loin. He went on with occasional hæmaturia and pain in the right, and later in the left loin, and lost flesh. A nodule appeared in the lower edge of the liver, which by the end of June extended two inches below the ribs. He became drowsy and unconscious, and sank. Post Mortem: All the viscera were healthy except the liver and kidneys. The right kidney formed a large irregular mass adherent to the liver. A large, firm, infiltrating growth occupied its upper half, growing round the adrenal, and changing its substance into caseous growth only recognizable as adrenal by its position and a little brownish pigment. Thence the mass grew upwards into the right lobe of the liver, and had spread from here through the liver substance as firm white

circular nodules. The lower part of the kidney was involved in growth but less regularly. This part was caseous, more like a scrofulous kidney. Pelvis dilated and sacculated. The ureter was dilated and thickened, and for its upper half its mucous membrane is covered by a vascular growth from its surface. This seemed to have begun half way down the canal, and growing thence had pushed upwards as a clot might do, forming a mould of the ureter at the upper part, and merely attached below. Bladder and other kidney normal. Microscopically : large round cells, some of them rather like epithelium — “a medullary cancer rather than sarcoma.” *Insp.* 1878, 263. *Med. Rep.* 251.

CASE 28. *Three months' history—universal extension.*—R. C., æt. 54 (m.). Admitted under Dr. Pye-Smith, April 7th, 1879, died April 22nd. Has had malaria and syphilis. Five years ago, on his way to work, he felt a sudden pain in the right hypochondrium, which sent him to bed and kept him there seven weeks. He was weak, but had no other symptoms. He has had a slight cough for five years, and has been sick now and then, generally in the early morning. Two months ago he began to have pain in the back and weakness, and for three months has had loss of appetite and flesh. Six weeks ago he gave up work. The pain is in the lumbar spine, epigastrium, and left hip. On admission—Fulness of right side of abdomen, swelling the size of the palm of the hand between ninth rib and navel. A nodule in the skin 2 inches above pubes. There is a solid mass in this position; pressure on it causes pain in the back. Liver normal. Lungs, signs of slight bronchitis. Urine 1025, acid, albumen, 30 ozs. daily. The veins on the left side of the chest and abdomen are the larger. The growth does not cross the mid-line, and has a definite lower edge at the navel. Pain and a good deal of vomiting continued. With a trocar some blood and large granular round cells were drawn off. This was followed by acute pain, especially in the right shoulder. The patient grew feebler, and his bronchitis worse. Died April 22nd. Post Mortem : Right pleura, some pleurisy. Right lung covered with nodules, mostly very small but some as big as peas or beans, most numerous in the superficial layers of the lung tissue. None in parietal or visceral pleura. Mediastinal glands much enlarged, especially in front of the left bronchus, where they formed a mass the size of a small apple. Abdomen, lymph and pus all over between the viscera. Liver, 90 ozs.; three or four large, several small, nodules; the biggest the size of an orange, this formed the tumour felt during life. All the larger growths are caseating and softening centrally. The lumbar and mesenteric glands, full of caseating growth, formed a large mass about the aorta. Each adrenal contained a minute nodule of growth. Right kidney, 5 ozs., normal, with a minute fibroma in one pyramid. Left kidney, 21½ ozs., nearly destroyed by what was clearly the primary growth. This occurred in lobulated masses, some of which were white and medullary, and growing into the little that remained of the renal tissue as a lobulated mass. It also projected into the interior of the pelvis as a firm lobulated growth, discoloured on the surface as if it must have bled during life. (No hæmaturia is known to have occurred.) One part of the growth formed a circumscribed encapsuled round mass, the size of a small apple. This was universally caseating, and was thought to be the starting point of the disease. Microscopically : the growth consisted of a closely interlaced mass of spindle cells and cells with long pointed processes. There was no trace of any alveolar arrange-

ment. The nuclei are not large, the cells not epithelioid. Acetic acid caused no increased opacity, but there was a good deal of granular matter present before it was added. *Insp.* 1879, 162. *Med. Rep. Clinical*, 240.

CASE 29. *Growth, aspiration, peritonitis. No metastases.*—F. S., æt 50 (m.). Admitted under Dr. Pavy, February 2nd, 1880; died February 6th. The history cannot be found, but on admission he had anæsthesia of one side of the face, paralysis and wasting of the left side of the tongue, and a large soft tumour in the right side of the abdomen. This was punctured on the 5th, and a dirty brown fluid drawn off. Pain in the abdomen immediately set in; he sank and died. Post Mortem: Brain normal. No disease affecting IX. or V. anywhere. Trunk of IX. in anterior triangle full-sized white and glistening. No note of XII. Left side of tongue contrasted markedly with right, it was flabby, pale, and thin. Microscopically: an excess of fat and connective tissue; the muscular fibres retained their striation, but one or two were granular. Lungs and heart normal. Liver normal. Left kidney 6 oz., pale and fatty. An acute suppurative peritonitis with creamy yellow pus. Colon adherent to mass and pushed down by it. The right kidney formed a large cystic tumour the size of an adult's head, projecting forwards. Part of its anterior wall was free, and no doubt after puncture some of its contents had escaped into the abdomen. But to the right side it adhered to the aorta, and behind it was an abscess in the iliacus extending towards the groin. The cyst held two pints of red-brown fluid, fœtid, with the look and smell of an unencysted hepatic abscess. Wall rough and thick. Growing from its upper part was a soft flocculent mass of new growth, on section almost encephaloid, emitting a creamy juice, discoloured and sloughing on the surface. Ureters and bladder normal. Microscopically: this was a true carcinoma, cells large and of varying shape, with immense nuclei, often two or three, and large nucleoli. There was a distinct alveolar structure. *Insp.* 1880, 67.

CASE 30. *Congenital sarcoma of adrenal, deposits in skull and skin.*—F. B., æt. 6 months (m.). Admitted under Dr. Pye-Smith, August 25th, 1881, and died September 25th, 1881. The infant was admitted with swellings in the head, left arm, and leg. The parents were healthy and the child born at full term. When he was 2 months old a lump appeared on the posterior fontanelle which has grown quickly since, and is now the size of an orange. soft and fluctuant. Other lumps have appeared and grown rapidly since. The swellings increased in size and the child lost strength. One mass destroyed the right eye, others appeared on the legs and elsewhere. Post Mortem: the tumours on the calvarium were situated in the bone and projected both outwards and inwards, there were three, one described above, and one in each frontal bone. They were soft and full of blood, the inner and outer halves of each were separated by a thin worm-eaten layer of the original bone. Dura free. Brain, spine, and lungs normal. A growth in the posterior bronchial glands. Liver, 9½ oz., contained six patches of growth 1½-in. by 1½-in. across. Some mesenteric glands a little enlarged. Adrenals both replaced by a soft vascular growth. With the exception of a projecting hæmatoma on the left side they retained their normal shape, but measured about 4-in. by 3-in. each. Kidneys normal. Left arm, a nodule 1-in. across in the subcutaneous tissue. Right leg, a similar larger one. Right thigh, one 2½-in. long, lying in a cavity in the muscle, of

which the bottom was formed by thickened periosteum. Ribs 5 and 6 were infiltrated about the centre for some 2-in. by a soft growth containing bony spicules. The growth was a small round celled vascular sarcoma.

Insp. 1881, 289. *Med. Rep.* 71.

CASE 31. *Signs of bronchitis six months, growth found post mortem, locally infiltrating, 8 inches long.*—P. S., æt. 55 (m.). Admitted under Dr. Fagge, June 1st, 1881, and died July 1st. A porter, used to lifting heavy weights. He complains of cough and wasting for the last five months, with night sweats. No hæmoptysis. There are the physical signs of bronchitis, with wasting and pyrexia. Diagnosis, phthisis. Urine normal. Temperature, from 102° or 103° to 99·6°. He gradually sank and died. Post Mortem: Lungs emphysematous, œdema. No phthisis, scarcely any bronchitis. Heart, brown atrophy. Peritoneum contained fluid. Stomach and intestines normal. Liver atrophic. Spleen shrunken. Right kidney and adrenal normal. Left kidney, an oval mass, 8 inches long, retained the shape of the organ. A good deal of the upper end was healthy, the lower part was occupied by some bosses of malignant growth which formed rounded tumours the size of three or four small apples, partly caseating, partly fibrous, clearly the primary growths. They grew into the kidney pelvis projecting from the mucosa as flat nodules with depressed centres the size of a shilling. Ureter free. Calyces scarcely dilated. In front was a large mass of glands infiltrated with growth, which was softer than that in the kidney, white and homogeneous, not encephaloid. Growth extended into the left psoas. The large veins were compressed, but contained no clot or extension of growth. Microscopically a sarcoma, large round or oval cells with vesicular nuclei, a few with two nuclei. These in parts formed the great bulk of the tumour, embedded in a granular and fibrillated matrix. In other places there is much fibrous tissue. Some spindle cells here and there. The growth has considerable tenacity. *Insp.* 1881, 198.

CASE 32. *Tumour three months, metastasis in lungs.*—J. G., æt. 64 (m.). Admitted under Dr. Fagge, July 28th, 1881, and died September 20th. Nine months ago pleurisy left side. Three months before admission a bale of wool fell on his left side, and on the second day after he noticed a tumour there. He had a good deal of pain, increased on movement. The mass increased and filled the left side of the abdomen, the patient got thinner and gradually sank. Urine contained a few pus cells, otherwise normal. Post Mortem: Three patches of growth the size of a shilling in right pleura. Three nodules right upper lobe. Left lung studded with hard grey nodules. Left side of abdomen full of growth, over which passed the descending colon. The rest of the intestines lay to the right. The mass was lightly adherent over 3 inches to the anterior abdominal wall. Liver, 54 ozs., fatty. No glands noted. Right kidney normal. Left kidney, 7½ lbs. All the kidney left was a piece half the size of a small orange. Growing from this was the large tumour, the upper part solid, irregular, bossy, white; the lower lacerable and red. *Insp.* 1881, 284.

CASE 33. *Large growth, tumour one year, hardly any metastases or invasion.*—R. B., æt. 49 (m.). Admitted under Dr. Moxon, March 10th, 1881, and died March 25th. Family and personal history good. A year ago he had pain

in the left loin and noticed a mass the size of the palm of his hand. The pain was intermittent, at times severe. Two months ago hæmaturia—he passed about half-a-pint of blood in two days. On admission, blanched, wasted, œdema of both legs (especially the left), which has been present some five months. Superficial abdominal veins dilated. A tumour fills the left and central part of the abdomen, it is round, hard, solid, and dull. Lungs normal. Urine normal. He was relieved from pain by morphia and died in a fortnight. Post Mortem: one enlarged gland in lower end of mediastinum. A few mesenteric glands slightly enlarged but no definite growth in them. All the large veins free, lungs and liver free, lumbar glands not mentioned. In the other kidney several white nodules from half-a-pea downwards. They were softer than the growth on the other side, and yielded a small amount of milky juice, the remaining structure was pale but healthy. Bladder and testes normal. An enormous mass in left side of abdomen, the colon ran as a small compressed tube vertically down the median line. The tumour was very elastic and had a dense tunica-albuginea-like capsule. Pancreas spread out and flattened in front of it, adrenal behind it. Neither was invaded. Vessels enlarged but normal. Ureter normal but blocked by a small uric acid calculus. The lower part of the kidney was healthy, and rose so abruptly from the mass that the tumour seemed extrarenal, but careful examination showed kidney substance spread out over the lower end of the tumour, which had grown in its head. On section blood clot and a tough yellow fleshy material, which microscopically consisted of a tough fibrous material with a number of leucocytes in the spare juice. *Insp.* 1881, 91. *Med. Rep.* 286.

CASE 34. *Growth of kidney and adrenal—2½ years' history—local invasion—metastases in intercostal space.*—T. R., æt. 37 (m.). Admitted under Dr. Willis, December 13th, 1862, and died July 29th, 1883. Two years before admission, and ten weeks after recovery from rheumatism, he began to have attacks of pain in left hypochondrium which used to cause him to take to his bed at intervals of three or four weeks. In October, 1881, *i.e.*, fourteen months ago, he suddenly passed ½-pint of blood, and went to hospital for four months, during the first fortnight of which he used to pass blood and clots. This has not recurred since. He got better and went to Dover for three months. He then began to vomit half-an-hour after his meals, to have the pain constantly, and to lose flesh. Six weeks ago he developed worse pain in the abdomen, groin, and root of the penis. On admission a tumour, noticed last month, filled his abdomen from ribs to ilium, it was tender, dull, solid, smooth, and presented a notch anteriorly. The urine was normal, now and always. During his stay the mass did not markedly increase, the pain and sickness varied. Post Mortem: In the right intercostal space close to sternum was a mass of fleshy, partly caseous growth, 1-in. by ½-in. Lungs normal. The stomach hardly more than a tube, passing vertically down over the mass, and then bending sharply to the pylorus. Liver normal. Pancreas flattened. Mesenteric glands normal. Several lumbar glands infiltrated. Right kidney normal. The left kidney formed a large lobular mass, and filled the left side of the abdomen from diaphragm to pelvis. The stomach and colon ran down vertically in front of it, the pancreas imbedded in its anterior surface. Everything in front of the twelfth dorsal and first lumbar vertebræ was infiltrated, but the bones were unaffected. The vena cava was displaced and adherent.

its wall puckered by infiltration. Left renal vein blocked by a tongue of growth which grew into it and projected from it into the cava. The muscles behind were infiltrated, and near the spine the tissues were yellow and gummatous. The mass itself was in two parts, one yellow, firm, elastic, into which the ureter ran—clearly the kidney, but without any renal tissue. The other softer, containing much blood and pigment, apparently a larger adrenal. No other adrenal was found this side. Microscopically the structure was the same, much of it fatty, the rest like a myxoma, but on careful examination round and oval nuclei were seen contained in large irregular cells of extreme delicacy, whose outlines were hardly visible. Nothing definitely cancerous about it, it looked like a sarcoma with mucoid degeneration. The right adrenal contained a large mass of fleshy growth very similar microscopically *Insp.* 1883, 262.

CASE 35. *Five years' history—nephrectomy—shock—a few nodules in lungs and liver.*—M. W., æt. 54 (f.). Admitted under Dr. Moxon, on October 1st, 1883; and died November 12th. The patient had had hæmaturia for five years, and a tumour had been felt in her abdomen since 1880. She was admitted with a large movable mass in the right loin, from which a little blood was obtained by puncture. The urine contained blood and pus. November 11.—Nephrectomy by Mr. Jacobson under chloroform and ether, lasting $1\frac{1}{2}$ hours. She became cold, pulseless, semi-conscious, and died. Post Mortem: The lungs contained two or three nodules of a firm, soft, white growth. One was 1 inch across. They contained epithelioid cells of various shapes. In the vena cava lay a little growth-like clot, which was considered innocent. The ligatures were satisfactory, and the peritoneum was shut off from the wound. There was one small nodule in the liver. Adrenals normal. Left kidney, uterus, etc., normal. Right kidney: A large nodular mass occupied two-thirds of it, in greater part caseating, the edge formed by fungating growth. The rest of the organ was swollen and oedematous-looking, as it is when the vein is obstructed. Pelvis dilated. On subsequent examination, Mr. Jacobson found the renal vein completely blocked and invaded by growth, which, however, had not gone far enough to in any way hamper the operation. *Insp.* 1883, 394.

CASE 36. *Growth of adrenal, direct extension to mediastinum, 3 months' history.*—J. W., æt. 32 (m.). Admitted under Dr. Goodhart, January 5th, 1885, and died January 31st. Three months ago he began to feel ill, lost his appetite, and had swelling of the feet. Seven weeks ago he fell so ill that he took to bed and has stayed there since. The last week or so he has passed little urine and that thick and high coloured. On admission, weak and wasted. Urine normal. Some oedema of ankles and wrists. No pain. Tenderness on pressure over left kidney. The oedema gradually subsided. In a fortnight's time he developed pleurisy at the right base. A week later 52 ozs. of purulent fluid were withdrawn, and he died a few days later. Post Mortem: Left pleura thickly coated and lung compressed. Right side healthy. Glands in posterior mediastinum malignant, hard, and white. Growth had spread along bronchi into lungs. Nowhere had it ulcerated into the bronchi. There was an irregular mass at the bifurcation of the trachea. Largest gland the size of a pigeon's egg. Right adrenal normal. Left adrenal, 20 ozs., wholly converted

into a malignant mass retaining its original shape. It had not infiltrated the tissues round or compressed veins. It was retroperitoneal and pushed the pancreas forward. Most of it was soft and breaking down. There were occasionally hard whitish nodules, with here and there a peculiar yellow tint like that of the vanished adrenal. The growth could easily be shelled out from the surrounding tissues—it adhered to the top of the kidney. It had grown in the direction of least resistance—up and out. The œsophagus was implicated from the bifurcation of the trachea down. The interior was ulcerated, and this entered the lung, forming a sac the size of a Tangerine with malignant walls leading from the œsophagus. Nothing else abnormal. Glands, liver, and lungs free. *Insp.* 1885, 43.

CASE 37. *Seven years' hæmaturia, no metastases.*—S. H., æt. 60 (m.). Admitted under Mr. Durham, August 18th, 1885, and died August 29th. In 1878 he first noticed bleeding from the penis. He had retention, the passage of a catheter was ineffectual, but a warm injection brought away a quantity of clotted blood and urine. In 1880 he had a similar attack and was treated two or three weeks. Some time afterwards Dr. Foster noticed enlargement of the right side of his abdomen. In July, 1885, pain came on and lasted three weeks, and since then there has been no blood noticed. On admission, a mass in right loin moving on respiration; dull. Its limits are 1 inch below ribs, 1 inch to right of umbilicus, $\frac{1}{2}$ -inch below umbilicus. No fluctuation. Veins are more distinct on this side of the chest and abdomen. Urine 1020, acid, a trace of blood. August 21st, operation; oblique incision. Large perirenal veins. Kidney punctured with a trocar. No fluid escaped, wound closed. The urine became ammoniacal, and the patient died in a week. Post Mortem: No peritonitis. Right kidney, 53 ozs., firm and hard. Renal vein tortuous and dilated. Posterior part of kidney adherent round wound. On section no kidney tissue remained, the upper part was yellow and caseating, with oily masses, the lower was tougher, and showed cysts filled with recent hæmorrhage. Into this mass the ureter opened freely. Mesenteric and lumbar glands, lungs, liver, other kidney, and all other organs healthy and free from metastases. *Insp.* 1885, 270. *Surg. Rep.* 111, 36.

CASE 38. *Cerebral hæmorrhage, growth of kidney found post mortem.* H. H., æt. 55 (m.). Admitted under Dr. Taylor, November 19th, 1885; died December 3rd. He was admitted recovering from a sudden loss of consciousness. He remained drowsy and had headache. Then he had one or two fits, became comatose, and died. Urine, 1018, normal. Post Mortem: Extensive cerebral hæmorrhage. Left kidney: upper half converted into a white malignant growth sharply defined from the healthy part. There were one or two minute nodules in the perirenal tissue. No healthy kidney tissue at the upper end. There were two nodules of growth in the lower end of the organ the size of marbles. Pelvis normal. Glands, lungs, liver, &c., normal. *Insp.* 1885, 389.

CASE 39. *Passage of growth by vena cava to heart. Tumour 4 years, growth and death in three months after miscarriage.* E. T., æt. 25 (f.). Admitted under Mr. Howse January 26th, 1886; died March 2nd.—Never very healthy; apparently anæmic and tuberculous. Three children alive; eldest 4. On December 18th a miscarriage, preceded by a month's bleeding from vagina. Ever since then she

has been in bed. Every attempt to get up brings on bleeding. Loss of flesh, weakness, bad appetite. Two or three times difficulty in micturition. For four years she has noticed a swelling in the right side of her abdomen. Originally the size of an egg, it grew gradually at first, more rapidly since her miscarriage, and is accompanied by a little pain; more latterly. On admission ill and weak. A solid, smooth, hard mass in right side of abdomen, reaching $1\frac{1}{2}$ inches to left of umbilicus. Resonance between this and the liver. The mass itself dull. Urine 1027; albumen, blood. Temperature 100—103°. Dr. Hale White diagnosed sarcoma of the kidney. Dr. Horrocks found the tumour was not uterine. Intermittent hæmaturia was present for a month. Operation, March 2nd; anterior incision. The patient died during the separation of the growth. Post Mortem: Lungs studded with minute growths, of which even the smallest are soft and creamy. Mediastinal glands full of growth. Right ventricle contained grumous delicate clots, suggesting growth, and similar masses were present in the vena cava and renal vein. Mr. Howse suggests that in the course of his manipulations he must have caused some of this very soft growth to pass into the cava, and so produce this embolism. The mass in the heart is microscopically identical with that in the kidney. The liver contained several small secondary nodules, the largest $\frac{1}{2}$ in. across. One or two lumbar glands much enlarged by soft creamy growth. Left kidney large, healthy. Right kidney still retained its normal shape, and at the upper end its normal appearance, the whole forming a mass 9 by 6, with a fibrous loculated basis. The greater part was composed of a soft, cream-like substance, which flowed out and left a flaccid stroma. The renal vein was full of stuff of this sort, and when washed away the wall was covered with fibrous shreds which seemed to be the stroma of the growth. There were two or three isolated nodules in the lining membrane of the vena cava. Microscopically, the growth consisted of a large granular epithelial type of cell with many nuclei. The kidney had been separated from its bed as far as the hilum, and presented a pedicle capable of being dealt with by ligature. *Insp.* 1886, 82. *Surg. Rep.* 121, 26. *Specimen in museum.*

CASE 40. *Growth of kidney and adrenals, invasion of skin and lungs.*—R. E., æt. 46 (m.). Admitted under Dr. Pitt, November 13th, 1888, and died December 13th. Six months ago he had pains in right hypochondrium, shooting to the left shoulder and down both legs to the feet, often followed by a short attack of jaundice and dark urine. Four days ago the dark urine was clotted in the morning. He has lost flesh six months, and in the last three weeks little hard tender nodules have appeared beneath his skin. The pain has been less lately. On admission, he cannot lie on the right side. Several subcutaneous nodules, movable, are present on scalp, neck, arm, back, and leg. The abdomen is tender, and there is a round hard mass in the right loin, which ends 2 inches to right of navel and reaches $\frac{1}{2}$ inch below it. It moves independently of the liver. There is a sulcus, which is resonant, between it and the ribs above and in front. Urine normal during his stay. November 26th, Mr. Jacobson removed a nodule from the scalp, which proved to be a scirrhous carcinoma with a moderate amount of stroma, not very hard. November 29th, he passed into a condition of mental weakness, which lasted till death. Post Mortem: No pigmentation. Cranial bones not invaded. Brain normal. Lungs, tubercle both apices (microscopically). Secondary

deposits from the size of a pea to that of a pigeon's egg. Secondary nodules one in the pericardium, one in the liver. The peritoneum, abdominal and thoracic glands, and all other organs free. Both adrenals entirely occupied by growth, the left larger, though the growth was in right kidney. No normal tissue left, possibly the growth was primary here. Left kidney normal. Right kidney anterior half normal. Posterior half replaced by a mass of growth as big as the kidney, which projects beyond its upper end. It is contained within the capsule, and breaks through it at two places only. It is composed of soft rounded areas, separated by fibrous bands of compressed kidney tissue. There is an intermuscular growth in the loin, the size of a pigeon's egg. Ureter normal. The growth is in all situations a carcinoma varying from schirrus to encephaloid. *Insp.* 1888, 446. *Med. Rep.* 250.

CASE 41. *Pain five months—exploration—death—extensive glandular infection.*—P. D., æt. 48 (m.). Admitted under Mr. Lucas, February 28th, 1889 and died March 10th. Eighteen months ago he had a fall on his back, and has since had several attacks of lumbago. Four and a-half months ago he had severe pain in the right loin, and has been invalided since. Hæmaturia several times. Occasional passage of long clots. On admission: No hæmaturia; patient pale and sallow. In right hypochondrium and loin a smooth, hard, solid tumour, surrounded by resonance, except where it joins the liver. Flank dull. Mass movable and separate from the liver, which is enlarged, smooth, and tender. Abdomen rigid; veins distended. Bad entry right lung; râles; bronchial breathing. Left lung not so bad. Tenderness along spine, not localized. Operation, March 4th: Kidney explored, two or three glands excised and others felt on the posterior wall of the abdomen; wound closed. He sank and died in six days. Post Mortem: Spine eroded in upper dorsal region by extension of growth from lung. All along the thoracic spine were deposits in the glands over the heads of the ribs. Some pleuritic effusion right side, Lungs both studded with nodules of growth, chiefly in the pleura, but also in the lung substance along the vessels. Mediastinal and bronchial glands infected. Vena cava thrombosed from opposite the renal vein down to the junction of the common iliacs, and thence into the left common and internal iliacs. The thrombus did not fill the vessel, and was covered with p.m. clot. Large mass of glands behind duodenum. Liver stuffed with secondary nodules, firm. No deposits in portal fissure. Lumbar glands converted into large masses of growth. Left kidney: one small deposit. Right kidney nearly all converted into a mass of growth which fungated into the pelvis, but did not block the ureter. All round it deposits in glands and adjacent tissue. Renal vein full of growth which spreads along it to vena cava. Small secondary deposits in right ureter 3 inches from pelvis. Microscopically both growths consisted of small alveoli, filled with large spheroidal epithelial cells. *Insp.* 1889, 103. *Surg. Rep.* 56.

CASE 42. *Five weeks' hæmaturia, nephrectomy, death, invasion of vena cava and lungs.*—J. W., æt. 17 (m.). Admitted under Mr. Lucas, November 20th, 1890, and died November 30th. Seven weeks ago he fell 8 feet and hurt his left side. Previously to this he had for some time had a dull aching pain in the left hypochondrium. Five weeks ago he began to pass blood; this lasted nine days, intermitted for a week, recurred, and has been present since, unaffected

by rest and milk diet. Rigors and loss of flesh. On admission: Firm smooth tumour in left loin and hypochondrium, resonant in front, not fixed, not fluctuating, reaching to umbilicus, and 1 inch from mid-line. Urine 1030, acid, blood, urea about 300 grains. Operation, November 28th: Anterior incision. Kidney adherent, covered with large veins. Nephrectomy. Counteropening and drainage. Chloroform was given for 1 hour 50 minutes. The patient was restless and sick, and died two days later. The kidney removed weighed 2 lbs. 4 ozs. and measured $7 \times 4\frac{1}{2}$ inches. It was nodular on the surface. On section soft, hæmorrhagic, degenerate in patches. Post Mortem: A few small nodules in both lungs and pleuræ. Adrenals and liver normal. Left renal vein was filled, from the ligature to the vena cava, with a thrombus composed of growth covered with clot. There was a large irregular polypoid mass protruding into the vena cava, which at first looked like clot, but microscopically was composed of very large cells filled with fatty granules, resembling epithelial cells in size and shape. No blood or peritonitis in the abdomen. In the pedicle and round it were some glands containing secondary deposits, and masses of growth which might have been extensions from the renal tumour. Right kidney, 5 ozs., normal. Bladder normal. *Insp.* 1890, 462. *Surg. Rep.* 166A.

CASE 43. *General glandular invasion, lungs free.*—M. Y., æt. 62 (m.). Admitted under Dr. Taylor, July 16th, 1890, and died January 18th, 1891. Admitted for epigastric pain and wasting. Two years ago he had epigastric pain, worse after meals, usually aching, rarely shooting in character. A year ago he got worse, has been invalided since, and in bed since February, with loss of flesh; no sickness or flatulence. For the last 18 months he has had shooting pains from his buttocks to his feet. On admission: He has moderately enlarged glands in the left axilla and the groins. He is suffering from "Tar Molluscum." His abdomen is retracted and resonant. Liver dulness extends to $\frac{1}{2}$ inch from the navel. Lungs normal. Urine, 1010, normal. Fæces sometimes colourless. During his stay he had variable pain, and tenderness is once recorded at a spot at the outer border of the erector, $1\frac{1}{2}$ inches above the iliac crest. He was sometimes sick, and sometimes had white stools. On the whole he gained weight (rising from 95 lbs. in September to 99 lbs. in December), but lost strength. Repeated attempts failed to detect any abnormal mass in the abdomen. Three weeks before death he passed blood and casts for the first time. Post Mortem: Axillary glands enlarged. Pleurisy left side. Lungs and thoracic glands free from growth. Liver contains two deposits the size of peas, and the glands in the portal fissure are infected. Adrenals, spleen, left kidney, normal. The mesenteric glands round the head of the pancreas are infected, and are the size of pigeon's eggs. The pancreas itself is half its natural size, but otherwise both gland and duct look healthy and contain no growth. The right kidney contains a growth the size of a cricket ball in its centre, which is degenerating. Microscopically: The growth is scirrhous carcinoma with spheroidal cells. The deposits in the axillary and mesenteric glands show more epithelial cells and less stroma. *Insp.* 1891 26. *Med. Rep.* 280.

CASE 44. *Sarcoma of adrenal—mycosis fungoides—no metastases.*—J. B., æt. 46 (m.). Admitted under Dr. Pye-Smith, January 17th, 1891, and died

March 30th. Personal and family history good, no syphilis. A year ago he was treated for itching all over. In July the sore on the right shoulder appeared and was followed by those on the left scapula and right loin. On admission, lungs, heart, and urine normal. On the right eyelid is a mass the size of a filbert, of which the surface is granulating. All over the chest the skin is covered with dry epidermis. On the spine of the left scapula is an inflamed patch 5 ins. across, raised, with a central depression, which has vertical edges and is filled with slough. There is a granulating patch on the right shoulder, and a few raw patches on the outer side of the right leg. On both surfaces of both arms the skin is dry and pigmented in patches, in some places there is scabbing. Over the right loin is a large swelling, the skin round it is inflamed, and there is a patch of slough on its lower and outer part. Diagnosis: *Mycosis fungoides*. February 11th.—All the raw patches decreasing in size. They give rise to a copious serous exudation. March 6th.—The place on the left shoulder has nearly disappeared, that on the right has increased a little, and is now 4 ins. by 5 ins., and raised $1\frac{1}{2}$ in. from the surface. March 10th.—Chronic acid injected into mass on loin. March 19th.—The patch over the eye is worse, the left forearm is nearly well, the right arm is raw from the shoulder to the elbow, a new patch has appeared below left scapula and two on the left arm. March 26th.—Sinking. Right arm better, left worse, back the same. March 30th.—Died. Post Mortem: Tumours as noted above. Brain and spine normal. Pleuræ adherent, hypostatic pneumonia, and at the base of the right lung a gangrenous patch; adherent pericardium. Liver, 89 ozs., fatty and cirrhotic. Capsulitis of spleen. Left adrenal normal. Right adrenal, $4\frac{1}{2}$ -ozs., enlarged by a fleshy growth which quite obscured the normal structure. It was vascular, with a few caseating patches and measured $2\frac{1}{2}$ ins. by $1\frac{1}{2}$ ins. Kidneys, bladder, testes, and all else normal. The tumour of the back and adrenal were histologically the same. They consisted of an aggregation of small round cells quite uniform in size and shape, with little stroma. They did not seem to differ at all from small round-celled sarcoma. *Insp.* 1891, 107. *Med. Rep.* 377.

CASE 45. *About a year's duration; hæmaturia 6 months; some minute metastases in lungs.*—J. J. æt. 43 (m.). Admitted under Dr. Goodhart, March 28th, 1891, and died April 26th. Admitted for hæmaturia and wasting. Good history, personal and family. In February, 1890, he had violent pains in the epigastrium which laid him up for a month. They came on suddenly and made him sick. In October he noticed his urine dark, with clots, and he had pain. He recovered, went to work, and in a few weeks had recurrence. Hæmaturia has been present off and on since. A month ago he had congestion of the lungs. He thinks he has been losing flesh a year; more rapidly the last six months. On admission: Right lung normal, left lung dull from fourth rib down, with a rub. Systolic bruit. Urine, acid, 1025, blood and pus. During his stay he had several rigors and rises of temperature. Some bloody serum was drawn off from his chest, and he showed signs of a mass in the abdomen, though the palpation of it is not noted. Post Mortem: Lungs, three nodules of growth, average size of a pea. On the right side recent pleurisy and broncho-pneumonia. On the left side the lower lobe is airless from compression. Thoracic glands normal. There was a large mass in the upper left abdomen; spleen pushed up, colon pushed forward and collapsed but not

compressed. The lower internal edge reached the middle line as low as the bifurcation of the aorta. Liver, spleen, and lymphatic glands normal. Stomach, pancreas and spleen lightly adherent. Adrenals—Right, normal; left, absorbed in mass. Right kidney normal. Left kidney: In upper part was a cavity holding grumous mass and clot; in the middle is a white growth, partially encapsuled; lower end normal. Microscopically: sarcoma with some long spindle cells, the fibrous arrangement often being concentric round the blood vessels. *Insp.* 1891, 146. *Med. Rep.* 341.

CASE 46. *Tumour 18 months, nephrectomy, shock, one gland infected.*—A. W., æt. 29 (f.). Admitted under Dr. Shaw, May 30th, 1891, and died June 15th. Five years ago she used to have a variable aching pain in the right side, under the lower ribs, while doing her work as housemaid. Three years ago she married, and has two children. She had a miscarriage (at three months) six weeks ago. She has noticed the present tumour for 18 months. The pain, which has always been localized, is worse lately. No hæmaturia. There is an irregular, smooth, fairly movable mass in the right half of the abdomen. Its long diameter is oblique, running from just below and to the left of the ensiform cartilage to just above and behind the right anterior superior iliac spine—above it is prominent and cystic. Nothing else, abnormal. Urine normal. June 8th: Aspirated; blood drawn off—no cells seen. June 18th: Operation; nephrectomy; followed by death in two hours. Post Mortem: Lungs and heart normal. The cavity left by the nephrectomy was found in a satisfactory state, but at the upper end was a nodule the size of a hen's egg adherent to the adrenal. This was the pedicle of the tumour, and not a secondary deposit. One gland beside the aorta was infected. Adrenals, left kidney, liver, and other structures normal. Microscopically the growth is a round-celled sarcoma. The Museum catalogue thus describes the affected kidney:—A right kidney, at the inner margin of which is situated an oval tumour $3\frac{1}{2}$ inches in its longest diameter. It is closely applied to the hilum of the kidney, and the renal vein crosses its anterior surface. The kidney has been laterally compressed and moulded to the convexity of the tumour. Anteriorly the growth is nodular, some of the nodules projecting into the dilated renal vein. On the back the surface is rough and was firmly adherent. On section the growth is composed of homogeneous material traversed by strands of translucent fibrous tissue. Histologically it has the structure of a spheroidal-celled carcinoma with abundant stroma (c.f. diagnosis above). *Insp.* 1891, 220. *Med. Rep. Clinical*, 338.

CASE 47. *Sarcoma in a child, invading liver and pulmonary artery.*—T. M., æt. 5 (m.). Admitted under Mr. Durham, November, 1892, and died January 4th, 1893. Three months ago he fell over a stool, and for a few days had pain in the left side. A fortnight later he began to lose flesh, and has continued to do so since. Ten days ago a tumour was observed in his abdomen. On admission: hard tense tumour in left side of abdomen. Liver normal. Urine normal. December 13th: Exploration with a needle; blood and a few masses of round cells, some containing pigment. The tumour continued to grow rapidly; the liver extended to $2\frac{1}{2}$ inches below the costal margin, superficial veins became distended, and the average excretion of urine sank from 11 ozs. to 4 ozs. Post Mortem: Lung—Small patches of collapse, probably pneumonia. Left pul-

monary artery filled by a mass of soft adherent growth, which contains no blood pigment. Left renal artery dilated. The vein admits a small finger, and contains adherent clots, softened centrally. Liver, 59 ozs. (normal is 19), reaches to umbilicus; contains many soft diffuent nodules. Right kidney normal. Left kidney: 54 ozs.; no renal tissue. Growth included in capsule, but breaking through it at a few points; soft, and caseating centrally. Ureter normal. Microscopically: In the liver, numerous nodules of round-celled sarcoma, composed of rapidly-growing cells, $8\ \mu$ in diameter. A few were ovoid, $24\ \mu$ by $8\ \mu$. They stained deeply, and were granular. The nucleus took up nearly the whole cell. Lungs showed only broncho-pneumonia. *Insp.* 1893, 11. *Surg. Rep.* 163, 48.

CASE 48. *Hæmaturia one year, œdema and ascites a few weeks, infection of lymph and blood channels.*—C. K., æt. 58 (m.). Admitted under Dr. Goodhart, April 28th, 1893, and died May 22nd. Admitted for wasting, weakness, and ascites. A year ago he noticed blood in his urine, and three weeks later had pain in his loins and passed more blood with clots. A few weeks before admission he noticed his legs swelling, and soon afterwards his abdomen. Urine has been scanty lately. On admission: Abdomen tense, abdominal veins dilated, liver slightly enlarged, urine 1014, a trace of albumen, (?) cirrhosis of liver. Ascites increased, and he died. Post Mortem: Pleura over the lower right lobe infiltrated, white lines showing course of infection along the lymphatics. Bronchial glands infected. Heart, ante mortem thrombus in right auricle continuous with that in vena cava. Upper half of vena cava swollen to twice its natural size, and full of clot, which looked malignant, and was softening centrally. The lower half is filled with hard adherent clot, which extends into the iliacs. Right renal vein thrombosed. 110 ozs. clear fluid in the peritoneum. Liver slightly enlarged, contains one secondary growth. Abdominal lymphatic glands affected. Left kidney normal, cortex cystic. Right kidney, twice normal size, irregular, soft, studded with masses of growth, no renal tissue apparent. Microscopically: Alveolar carcinoma, masses of cubical cells divided off by fibrous partitions. *Insp.* 1893, 187.

CASE 49. *Death from pneumonia at 8 months. Sarcoma found in adrenal.*—A. M., æt. 8 months (f.). Admitted under Dr. Perry (Clinical), August 13th, 1894, and died August 23rd. Admitted for cough and wasting of one month's duration. Temperature, 102° . Post Mortem: Pleurisy and broncho-pneumonia right side. Left adrenal normal. Right adrenal contained a white growth with hæmorrhages into it, the size of a large pea, which microscopically was found to be a round-celled sarcoma. Kidneys normal. *Insp.* 1894, 334.

CASE 50. *Six months' course, deposits in sternum, lungs, glands, liver.*—T. T., æt. 64 (m.). Admitted under Dr. Hale White, January 28th, 1895, and died March 23rd. He has been healthy, but many years ago he lost his memory for a year, and after this had a goitre which was seen and eventually removed by Sir James Paget. He has been quite well till September, 1894, when he had a pain in the left chest with cough and wasting, which have continued since. There is now a small lump on the third costal cartilage beside the sternum, fluctuating, and surrounded by a tender area. Puncture of this yielded blood and large multinuclear cells, bigger than lymph cells.

Pulsation was transmitted through the swelling. Lungs normal. Urine normal. Mr. Dunn considered there was an inoperable growth, probably primary in the sternum. The further course of the case was increased pain, bronchitis, pneumonia, the appearance of a mass below the left clavicle, swelling of left arm, and extension of the sternal swelling. Post Mortem: Sternum and third and fourth left costal cartilages eroded by soft red growth. Both lungs contain vascular nodules of growth, many cystic or broken down centrally. Liver, a few nodules. Some abdominal lymph glands infected. Adrenals partly destroyed by growth. Left kidney: Lower third occupied by mass of tough white growth, nearly as big as a normal kidney, soft in places. Above, an encapsuled nodule not broken down at all, ?adenoma of accessory adrenal. Right kidney: several deposits of tough white growth, also a small nodule rather like the adenoma in the other kidney. Microscopically an oval-celled sarcoma with caseous change. *Insp.* 1895, 95. *Med. Rep. Clinical*, 58.

CASE 51. *Hæmaturia 5 years, two explorations, ligature of ureter, death. Recent secondary deposits in pleura, no other metastases.*—A. E., æt. 54 (m.). Admitted under Mr. Lucas, October 8th, 1896, and died January 27th, 1897. Five or six years ago he had dull aching pain in loins, upper thighs, and testes. He then passed some blood in the urine and the attack ceased in 24 hours. Since then he has had similar attacks once or twice a year, lasting about a day. Two years ago the attacks became longer, and the blood more plentiful. In the last year he has had three or four more severe attacks. Twelve days ago he passed pure blood and was dependent on a catheter for five days, since then the urine has been normal. Urine acid, 1022, no blood or pus, a little albumen. He had pain over the 11th & 12th dorsal and 1st lumbar vertebræ (renal Head's area). He continued to have intermittent passage of blood, some thin clots up to $7\frac{1}{2}$ -ins. in length. Operation, November 10th.—Kidney large, hard, firm, and smooth. No enlarged glands felt, no calculus detected, no fluctuation, wound closed. The patient's condition remained the same. Urine averaged 50 to 60-ozs daily. It gradually became purulent and ammoniacal. Operation, January 27th.—Incision through the old scar. Kidney much enlarged, a hard nodular, whitish growth projected from it anteriorly. It was firmly adherent and the patient's condition was bad. The ureter was ligatured and the wound closed. Death followed in seven hours. Post Mortem; Left pleura, 10 small nodules $\frac{1}{8}$ -in. across. Lungs, liver, supra-renals, lymphatic glands, all other viscera examined and found free from growth. A few cysts in the left kidney. Right kidney, 31-ozs., a large irregular mass of hard growth, only slight traces of renal tissue. Microscopically, a sketch showing an alveolar arrangement. *Insp.* 1897, 34. *Surg. Rep.* 168.

CASE 52. *Rapid course, glandular and direct extension, blockage of ureter a month before death the first symptom, abnormal adrenal.*—E. O., æt. 54 (m.). Admitted under Dr. Pitt, January 11th, 1897, and died January 24th. He was healthy till December 20th, 1896, when he began to have difficulty in passing water, which lasted a fortnight. He could only pass small amounts and desired to do so very frequently. On December 29th he passed $\frac{1}{2}$ oz. of bloody urine on two occasions, this was his first and last hæmaturia. Sub-

sequently he suffered from pain and sleeplessness. On admission he was thin and anxious. There was a hard nodular tumour in the right side of the abdomen, separated from the liver by a band of resonance. It extended into the right loin and filled a space bounded by umbilicus, iliac crest, and liver. A transverse ridge crossed its lower part, and there was a tender area near the umbilicus. The hand could be carried beneath its left edge. Bad entry right base. Urine 1020, acid, albumen, pus. Microscopically: bloodstained fragments in the urine, (?growth.) After admission he went rapidly downhill, vomiting without relation to food, and sleeping badly. Post Mortem: One deposit in a mediastinal gland. Head of pancreas and submucosa of duodenum infiltrated. The colon, which passed over the front of the tumour, was small and adherent, but not infiltrated. In the right iliac fossa a growth, 3 inches across, due to the kidney tumour, and extending down as a large cord in the course of the right ureter. Secondary deposits in the mesenteric glands. Left kidney, $7\frac{1}{2}$ ozs., normal. Attached to its upper end was a piece of adrenal, and on examination it was clear that the adrenal tissue extended into the renal tissue, as sometimes happens with adrenal rests. The patch of adherent adrenal was not homogeneous, but consisted of separate flattened nodules, and appeared to be above the capsule, though, as section showed, some of it went beneath it. Right kidney occupied and almost wholly destroyed by a large white softish growth, which extended down the ureter and partly blocked it, producing hydronephrosis. At the lower and inner part of the organ the growth had penetrated the capsule and formed a large nodular mass, which infiltrated the head of the pancreas and appeared as a white substance beneath the mucosa of the second and third parts of the duodenum. Mucosa of bladder normal, but at the orifice of the right ureter was a sloughing growth the size of a Brussels sprout, and this was connected with the growth mentioned above. Histologically the growth was a round-celled sarcoma. *Insp.* 1897, 28. *Med. Rep. Clinical*, 44.

CASE 53. *Death from suppuration in the liver, "renal rest" forming part of adrenal tumour found post mortem.*—W. S., æt. 55 (m.). Admitted under Dr. Shaw. The patient died with suppurative cholecystitis and cholangitis, fistula into the duodenum, and liver abscess. The liver section gave rise to a suspicion of actinomycosis. Kidneys normal. In the right adrenal was a round soft yellow tumour the size of a fives ball, containing one or two hæmorrhages. Microscopically it showed some normal adrenal tissue containing numerous spaces filled with blood. Around this adrenal tissue, but separated from it by a thick and irregular capsule of fibrous tissue, was kidney tissue containing a large number of glomeruli. In places there were large numbers of glomeruli, and hardly any tubules, but a number of small round cells, and also large cells resembling in character the suprarenal cells. Dr. Bryant noticed several glomeruli, the inner surfaces of the capsules of which were lined by these cells. *Insp.* 1897, 95.

CASE 54. *Short course, local and glandular extension.*—T. G., æt. 62 (m.). Admitted under Dr. Goodhart, April 1st, 1897, and died April 29th. Admitted for cough and shortness of breath. He has lost flesh for the last 12 months. For three months has had cough with dyspnoea and occasional difficulty in swallowing. On admission: Resp. 28. Dulness and diminished breath sounds

from sixth rib down on right side. Edge of liver at navel, surface firm and rough. Some ascites. Enlarged abdominal veins. Large hard gland left side of neck. He grew worse and on April 12th had œdema of the right leg which subsequently spread to the other leg, scrotum, and body wall. Glands appeared in groins and over clavicle. Urine contained indican. Post Mortem: Growth in the cervical glands. 68 ozs. fluid in right pleura. Growths under pleura and in lungs. Mediastinal glands infected. White patches in muscle of left ventricle. 48 ozs. fluid in the abdomen. Right kidney much enlarged and nodular from protrusion of white masses of growth which extended in all directions to involve liver, vena cava, and retroperitoneal glands. On section the kidney was full of a soft white or grey vascular growth. Hardly any trace of renal tissue. The vena cava was invaded by growths which formed a layer on its inner wall for $4\frac{1}{2}$ inches below the hepatic fissure. Below this it was filled with a thrombus extending to the division of the iliacs. Retroperitoneal glands full of growth and lumbar vertebræ eroded. Pancreas normal. Secondary deposits liver and left kidney. *Insp.* 1897, 167.

CASE 55. *Rapid course, invasion by blood stream, no hæmaturia.*—J. McN., æt. 50 (m.). Admitted under Dr. Pitt, May 11th, 1898, and died May 13th. He has had malaria, syphilis, and pneumonia. In October last he fell and strained his right side and subsequently had pain for some days. In January, while convalescent from pneumonia, he had severe pain in the right side for a week, followed by pain down the inner side of the right thigh, and the whole of the opposite leg became swollen. A few days later the right leg did the same. He has been losing flesh since. In February he noticed a swelling of the abdomen. On admission: Thin, legs and right side of chest and abdomen œdematous. He always lies on his right side. A large smooth uniform dull mass fills the right loin, its edge runs from $1\frac{1}{4}$ inch below the navel to the ninth costal cartilage. It does not move with respiration. An exploring needle withdrew only blood. Urine 1022, a trace of albumen, no blood. Lungs: Bad entry right base, râles behind; rub in second space in front. He developed signs of pleurisy on the left side, and died May 13th. Post Mortem: Secondary growths throughout both lungs. Small masses of detached growth in right side of heart. No ascites. Abdominal lymphatic glands normal. Adrenals normal. Mass in liver $4 \times 2\frac{1}{2}$ inches, signs of recent hæmorrhage. Large tumour in situation of right kidney, consisting of growth in the capsule of the kidney. There is old hæmorrhage in the centre. Only a small piece of renal tissue remains at the lower end. The growth has invaded the vena cava, which is filled with ante mortem thrombus. Left kidney rather granular. No microscopical report. *Insp.* 1898, 191. *Med. Rep. Clinical*, 212.

CASE 56. *Calculus—nephrectomy—growth.*—J. C., æt. 30 (m.). Admitted under Mr. Lane, June 7th, 1898, and died June 24th. Previous history good. Fifteen years ago pain in right side and red urine. These attacks occurred about once a year for some time; they then became more frequent, and for the last five years have occurred once a month. In October 1897 he was operated on for calculus, at Newport. Four operations were performed and five stones removed. He was admitted to Guy's with a foul discharge from a large renal fistula. His general condition was good. Urine 1020, acid, albumen, pus, no blood; urea, 1.3 per cent. Operation, June 15th: Kidney enlarged, adherent,

full of pus; containing three stones the size of filberts. A large abscess extended up beneath ribs and diaphragm. The kidney was removed piecemeal, part of the pelvis was left. Afterwards he was depressed, vomited from time to time, and died nine days later. Urine averaged 16 ozs., urea 1·4 per cent. Post Mortem: The remains of the right kidney consisted of the pelvis, in which there was a mass of fungating growth, which Dr. Perry thought was certainly growth and not granulation tissue. Suppuration had extended upwards behind the peritoneum, and the fibrous tissue in the portal fissure was itself infiltrated with pus. Downwards it had extended to the pelvis, causing retro-peritoneal cellulitis, and there was pus in the rectovesical pouch, and a local peritonitis. The stomach and duodenum had adhesions to the liver and gall-bladder respectively. All other organs normal. The other kidney weighed 8 ozs. and was healthy. *Insp.* 1898, 243. *Surg. Rep.* 69.

CASE 57. *Calculus, pyonephrosis—epithelioma—lymphatic infection—4 months' course.*—E. L., æt. 48 (m.). Admitted under Dr Shaw, March 21st, 1899, and died May 21st. Cystitis 23 years ago: recovery. Otherwise well till January last when he felt vaguely ill and began to lose flesh. No hæmaturia. On admission: A large hard tumour in the right side, moving on respiration. Lower and inner edges distinct and hard, upper edge 1 inch below ribs. It extends into the loin, down to the iliac crest, inwards nearly to the mid-line. It is dull, but traversed by a vertical band of slight resonance. Complete dullness behind. Base of right lung compressed. Urine 1016, acid, nothing abnormal. Urea 1·7 per cent. On March 30th, there was a peritoneal rub and some fever. There was albumen in the urine; no blood. Tumour tender. On April 4th a discharge of pus in the urine began and lasted three days—it contained no tubercle bacilli. On the 10th the rub and pain had disappeared. He lost flesh (7 lbs. in a month) and had a recurrence of tenderness, but not any increase in the tumour. Pain and œdema of the feet and ankles developed, and he died May 21st. Post Mortem: No deposit in lungs or heart. Vena cava pressed on but not infiltrated. The liver contains soft yellow nodules up to 2 inches across fluid centrally. Abdominal lymphatic glands are enlarged. Right kidney bigger than two fists, upper end loosely adherent to the liver, and behind it an abscess containing $1\frac{1}{2}$ ozs. pus. The capsule is perforated here. Round it the kidney tissue is very thin. Inside a large branched calculus was found surrounded by pus, and filling the dilated pelvis and calyces. The lower half or more of the kidney is replaced by a soft, yellow growth which had bulged towards the spine and infiltrated the neighbouring lymphatics, and had pressed on and almost entirely occluded the vena cava, which below it was blocked by ante mortem thrombus. Microscopically: An epithelioma with a marked bird's nest arrangement of cells; it looked very much like epithelioma of the œsophagus, the cells being squamous in appearance. *Insp.* 1899, 161. *Med. Rep.* 109.

CASE 58. *Adrenal rest tumour, hæmaturia, cystotomy, nephrectomy—death from gastric ulcer.*—J. M., æt. 58 (m.). Admitted under Mr. Golding Bird, September 18th, 1899; re-admitted November 28th; died March 2nd, 1900. When 13 years old had a kick in the left loin, followed by 7-8 years incontinence. Mic-turition always frequent. Pain over sacrum last two years. In April, 1899, he

had hæmaturia—no injury—no pain. This persisted, with occasional difficult micturition from passage of clots. The attacks lasted 5-6 days, with intervals of freedom of about the same length. Some clots were the size and shape of a finger. On admission the left kidney seemed rather the larger, but a week later no difference could be felt. Urine 1020, acid; blood and pus present. On one occasion a mass of epithelial cells was found undergoing fatty degeneration. Operation, October 19th: Cystotomy. The only abnormality found was a small ulcer on the left of the trigone, which was scraped. The patient went out, but continued to have the same symptoms. He was re-admitted, and although the swelling of the left kidney, noted above, had not recurred, it was decided to explore this side. Operation, November 30th: Kidney found enormously enlarged and covered with dilated veins. The upper and anterior borders could not be reached, and at the lower end a large knob projected down into the pelvis. The patient's general condition was so bad that the wound was closed. Operation, February 20th: A vertical incision was made, 7 inches long, which was afterwards extended. The peritoneum was opened, and both kidneys explored. The left kidney, which was adherent behind and above, was with difficulty removed. Eleven days later the patient passed a quantity of blood and slough from the rectum, and died. Pathologist's report: This section shows a diffuse deposit of carcinoma. The cells are very clear, and in alveoli, the walls of which are composed of very fine fibrous tissue, the appearance suggests a suprarenal origin for the tumour. Microscopically the pedicle removed was free from growth. Post Mortem: Only a partial examination allowed. Abdominal wound healthy; some brick-red fluid, probably broken-down blood, along course of psoas. Pedicle firmly ligatured, and no growth could be seen extending into it. Right kidney and liver normal. Some blood in the intestines. Stomach contained two pints of clot. On the lesser curvature, within 4 cm. of the œsophagus, was an ulcer 7×5 cm., the edges rounded and thickened, the floor formed by the muscular coat. It looked like a chronic ulcer, and there was no infiltration of growth into it. In the centre of its floor was a minute opening leading directly into one of the vessels along the lesser curve of the stomach.

Surg. Rep. 509

CASE 59. *Growth of adrenal, no invasion or metastases, 4 years' history.*—E. S., æt. 30 (f.). Admitted under Dr. Perry, November 9th, 1900, and died November 27th. In 1896 she first felt pain in the side, where she feels it now. This has since been intermittent. Frequent menstruation began then and lasted till May, 1900, when the function ceased. In November, 1899, she had a sharp attack of pain in the right side, and saw a doctor who diagnosed a small ovarian tumour. In August she was told she had enlarged liver and pleuritic effusion. In September her chest was tapped and 2 ozs. blood-stained fluid withdrawn. She has since had more pain. She cannot lie on the right side. There is now dulness and absence of sounds from the fourth rib downwards, and a small dull area on the left side. Voice-sounds audible everywhere. No râles. The liver, hard, with a rounded edge, is felt to reach to $1\frac{1}{2}$ inches from the right iliac crest. In the region of the gall bladder is a small round tumour, separable from the liver, not moving on respiration, not painful, not connected with the pelvis. Per rectum a hard mass the size of a small apple on the anterior wall. Urine alkaline. November 19th: Liver

bigger. Temperature always normal. Swelling in right iliac fossa less definite. Red corpuscles 5,240,000. Hydatid discussed. November 26th: Mr. Fripp made a vertical incision at the outer border of the rectus. Liver normal. Midway between its anterior and posterior surfaces was a swelling whose surface was covered with large veins. It displaced the kidney downwards and the peritoneum went on to it straight off the liver. It had no pelvic attachments. The wound was packed to form adhesions. November 27th: Packing removed—mass incised—blood and pulpy material issued. The cavity was packed and death followed in a few hours. Post Mortem: Lungs, lower third compressed from below, emphysematous, no evidence of old pleurisy. The lungs contained one or two nodules of growth the size of a pea. The liver was pushed up and the kidney downwards by a large encapsuled growth between them, 26×21 cm. Above it was firmly adherent to the under surface of the right lobe of the liver, and the edge of this was thinned out over it in front so much as hardly to be felt even post mortem when the hand was passed from liver to growth. Although adherent there was no invasion of the liver, nor did it invade any neighbouring viscera. On section the capsule enclosed a soft red growth with an irregular firmer yellow mass in its centre. No trace of the right adrenal found, Dr. Fawcett thought that this was the origin of the tumour. Left adrenal normal. Both kidneys normal, the right displaced downwards. Microscopically: An extremely vascular growth—great part of it having been destroyed by blood extravasation. Where not destroyed it consists of a mass of cells with very little fibrous tissue between them. *Insp.* 1900, 420. *Med. Rep.* 389.

CASE 60. *Two months' course, deposits in skin, pleura, liver, glands.*—H. S., æt. 34 (m.). Admitted under Dr. Bryant, November 30th, 1900, and died December 23rd. A tinplate worker, always healthy. At the end of October, 1900, he began to have pain in the loins shooting down the thighs, which got worse. Three weeks later he took to his bed. He has lost weight and strength. The pains come on with no apparent cause. The right leg has become weaker than the left, and there is pain in the left foot. In the right deltoid is a swelling the size of a marble, soft and movable, which has been there three weeks. Another is present, loosely attached to eighth rib in the right axillary line (where the shears touched him), this is larger and of rather older date. First dorsal spine prominent and tender, fourth dorsal tender. Chief reference of pain is to first and second sacral, and the posterior third of the iliac crest. Nothing abnormal to be felt here. Knee-jerk increased. Ankle clonus present. Liver and spleen impalpable. Chest normal. Urine 1005, alkaline, nothing abnormal, urea 1.3 per cent. Diagnosis, sarcoma or subacute myelitis. At first the patient improved, and his legs gained power. On December 17th his liver was found enlarged, and this increased till death. An intention tremor of the hands was noticed, and he finally died with hiccough and delirium. Post Mortem: The lumps noticed in life proved to be secondary nodules between muscle and deep fascia, softening centrally. Spinal cord normal. Thyroid atrophied, a yellow fibrous patch (not growth) in each lobe. Lungs normal. Right pleura, nodules of growth from a pin's head to a pea; recent pleuritic adhesions. Recently extravasated blood in the pericardium. The pulmonary artery contained an ante mortem thrombus, derived probably from the auricle. Liver, 3091 gr., yellow, mottled. Right lobe

infiltrated and replaced, some small nodules the size of a pea. Left lobe infiltrated and softening. Pancreas and glands invaded. Right kidney, 584 gr., upper half occupied by a white mass 6 cm. across. This had invaded the perinephric tissue on the inner side; on the outer it was coated with renal tissue. Left kidney, several deposits the size of a pea. Adrenals normal. Dr. Fawcett considered the kidney growth undoubtedly primary. Microscopically: The kidney showed a mixed spindle and round-celled sarcoma. The liver was invaded by growth, which in parts where the tissue is not wholly replaced seems to run along the course of the hepatic veins and interlobular capillaries, the intralobular veins are in many cases filled with growth. The liver cells are cloudy and atrophic. *Insp.* 1900, 451. *Med. Rep.* 397.

CASE 61. *Carcinoma, nephrectomy, discharge.* H. B., æt. 55 (m.). Admitted under Mr. Jacobson, November 8th, 1901, and discharged January 15th, 1902. Six months ago he caught his foot in a hole while running, had a pain in his left side, and an hour later passed urine containing much blood. For some time he passed highly coloured urine, and had recurrences of pain. Three months ago a tumour appeared in his left side, and he occasionally passed clots, one 8 inches long and as thick as whipcord. He could not sleep on his right side, as a mass seemed to fall across. On admission: A movable tumour in left renal region. Skiagram showed nothing abnormal. The diagnosis lay between a latent calculus dislodged at the time of the accident, and blood calculus. Operation, November 22nd: Kidney found enlarged and low down. It was bossy with what was clearly malignant growth. The structures at the hilum were matted together and difficult to recognize. Clamps were applied to them. Near the origin of the renal vessels was a grey mass, probably growth. The whole was removed as completely as possible, and the patient made a good recovery. Mr. Targett has examined a section of the growth and thinks it is a degenerating carcinoma. *Surg. Rep.* 17.

CASE 62. *Papilloma of pelvis, nephrectomy, death from recurrence.*—H. K., æt. 53 (m.). Admitted under Mr. Symonds, July 25th, 1902, and discharged October 19th. Previous history good. Three years ago he noticed that his urine was very dark, and again a year later. In the last three months pain has begun to occur in the right loin, groin, and testes, and a fortnight ago he had difficulty in passing his water, caused by clots. On admission: Right side of abdomen rigid, in right loin a large tender trilobed mass. Hydrocele right side and pain in right testes and thigh. Operation, July 30th: Kidney on exposure dilated and contained much dark fluid, with which escaped pieces of papillomatous growth. Pelvis and ureter thickened and dilated, one gland enlarged. The whole kidney and upper end of ureter removed. The pelvis was found choked with papilloma, and hardly any kidney substance was left. The patient recovered and went on fairly well for a month. At the end of six weeks he complained of pain in the left loin, and a mass could be felt there, which was considered likely to be secondary deposit. He went gradually downhill, and was ultimately discharged to an infirmary where he died. Microscopically the growth was of a delicate papillary nature. There was only a small amount of cellular tissue at its base, and nothing to suggest malignancy. *Surg. Rep.* 328.

CASE 63. *Six years' pain, no hæmaturia, nephrectomy.*—S. W., æt. 36 (f.). Admitted under Mr. Jacobson, April 4th, 1902, and discharged May 16th. Twelve years ago a difficult labour, no other illness. Six years ago she began to have severe pain in the left loin and was for several weeks an in-patient at two London hospitals, where floating kidney and neuralgia were diagnosed. She was then treated for seven months for laceration of the cervix. Since then she has remained well till a fortnight ago, when she began to have severe intermittent pains in the back, increased by movement. On admission: Dyspepsia, general condition good. Pain limited to left inguinal and lumbar regions, coughing painless. A mass can be felt in the left loin at the level of the iliac crest, dull, tender, moving with respiration, and about 2-ins. by $\frac{3}{4}$ -in. in size. Urine normal, urea 1.5 per cent., leucocytes 9000. Diagnoses suggested were growth, and pyo-nephrosis with calculi. Operation, April 15th: Oblique incision, kidney enlarged with grey patches on the surface, no calculi found, ureter healthy, no thrombosis or glands. It weighed 15 ozs., and only a small part near the hilum remained normal. The microscopical section was unrecognizable. The patient made a good recovery. *Surg. Rep.* 186.

CASE 64. *Papilloma—15 months' hæmaturia, nephrectomy.*—G. Q., æt. 60 (m.). Admitted under Mr. Jacobson, November 23rd, 1902, and discharged January 1st, 1903. History good. Fifteen months ago he first noticed that his urine was sometimes tinged with blood, especially after exercise. A year ago he felt a weakness in his back, and nine months ago he began to have attacks of sharp, shooting pains below the right ribs. Eight months ago he had a fall, and after this the pain got worse and extended across the right iliac region and down to the testes. Two months ago he passed a clot 2 inches long, the thickness of a toothpick. On admission: He seems healthy, except when his pain is present. There is some tenderness of the right loin and rigidity of abdominal muscles. The right kidney is more easily felt than the left. He cannot sleep on the left side as a mass seems to fall across. Urine, 1024, acid; blood and pus present. December 3rd, operation: The kidney was punctured, incised, and explored with the finger. It bled freely. The kidney was clearly a damaged one, though neither calculi or growth could be demonstrated. The ureter was cut low down and the kidney removed. On examination it was found to have a villous mass at the beginning of the ureter lined by cubical epithelium. The epithelium also invaded the substance of the kidney. Mr. Targett considered the growth malignant. The patient made a good recovery and was discharged January 1st. *Surg. Rep.* 21.

CASE 65. *Congenital sarcoma of adrenal, infiltration of liver.*—W. M., æt. 2 weeks (m.). Admitted under Dr. Perry, January 17th, 1902, and died January 18th. The child's abdomen was noticed to be swollen two days after birth. It has since become bigger. There was anuria for the first two days, and a bullous rash over back and thighs since the first day. Legs, lower back, and abdomen œdematous. Abdomen distended: superficial veins running up from umbilicus. Upper and right half of abdomen dull. Lungs normal, Respiration 64. January 18th: Temperature 102° morning. 104° evening. The only urine obtained ($\frac{1}{2}$ oz.) contained no albumen. Post Mortem: Slight broncho-pneumonia both lungs. A little ascites. Liver much enlarged, yellow, mottled, infiltrated throughout with what looks like new growth. It weighs

1850 grms., and the capsule is normal. Glands in portal fissure are also infiltrated. Right adrenal normal. Left adrenal, a large tumour $2 \times 1\frac{1}{2}$ inches, is quite independent of the kidney. Alternate layers of creamy white and deep red. In the centre many little yellow gritty points. Right kidney normal. Left kidney attached to tumour. Microscopically the adrenal shows a superficial layer of normal adrenal cortex, enclosing a small round-celled very vascular sarcoma. In part the cells of the tumour are arranged round the edges of small masses of homogeneous substance of uncertain nature. The liver presents a structure similar to that of the adrenal tumour. Only slight remains of normal liver tissue can be found. *Insp.* 1902, 36. *Med. Rep. Clinical*, 68.

CASE 66. *Five months' course, extensive lymphatic and local invasion—blood invasion.*—A. M., æt. 33 (m.). Admitted under Dr. Hale-White, May 8th, 1902, and died, August 27th. Came in for swelling of the leg. History good. He was well till March last, when he began to feel ill and run down, and had pain in hips, groins, and thighs, these got worse and his feet were always cold. He has been laid up two weeks. On admission, temperature 101° right leg oedematous from foot to groin, veins on leg and abdomen distended, femoral vein a hard cord. Liver normal, chest normal, urine normal. Oedema and pain decreased. In the middle of June he lost power in his right leg, but retained sensation. At the end of June a little fluid was drawn off from his left pleura. Meanwhile pain and paralysis varied. About a month later there was oedema in the other thigh, the enlargement of veins and upward flow in them was marked, there was some fixed dulness in the left flank, and an enlarged and nodular liver reaching to within two inches of the navel. Carcinoma of the pylorus was discussed. In the course of August he developed paresis of both legs and loss of sphincter control, the liver increased in size, sensation became abnormal, and finally two days before death there was some blood in the urine. Post Mortem: A solid mass of growth surrounded the cavity of the pelvis and adhered to its inner wall. It compressed all the vessels and the sacral plexus, but had not invaded the bladder and prostate. The right common iliac was thrombosed at its upper part. Over the prominence of the sacrum and on either side was a solid mass of growth continuous with that in the pelvis, and extending up on either side of the vertebral column as far as the crura of the diaphragm. The vena cava was invaded and completely blocked by the growth, the aorta surrounded but not invaded. The mass was continuous with deposits in the lymphatic glands, and on the left side with the kidney, which was much enlarged. It extended into the spinal canal by the intervertebral foramina over the lower 9 cm. of its length, and for this distance there was a layer of growth on the anterior surface of the dura mater. The nerves of the lumbar plexus were completely surrounded. Sections of the spinal cord were normal to the naked eye. On cutting through the muscles of the back in the lumbar region, a secondary mass was discovered which had spread through from the mass in front of the vertebræ between the laminae. The lungs showed old adhesions and were studded with nodules of new growth almost all the size of peas. In the thorax was a tubular mass of growth $1\frac{1}{2}$ cm. broad along the side of the aorta continuous with that in the abdomen. Azygos vein thrombosed. Heart, 210 gr. A worm-shaped cylinder of white growth hung from the vena cava into the right auricle for 3 cm., and

another polypoid mass the shape of a gourd adhered to and projected out of the auricular appendix, this was 5 cm. long. Liver, 4320 gr., enormously enlarged by deposits. Spleen normal. Lumbar glands and those in the portal fissure infected. Adrenals normal. Right kidney normal. Left kidney, 710 gr., enlarged by a mass of white growth which nearly replaced the renal substance. It was firmer than that in the liver and was considered the primary source. The glands at the hilum were enlarged. There was acute cystitis. Microscopically: A carcinoma with alveoli of varying sizes and shapes, lined mostly by a short cubical epithelium, with comparatively little stroma. The growths in lungs, liver, and pelvis were similar, that in the lungs being rather more fibrous than the rest. *Insp.* 1902, 375. *Med. Rep.* 173.

CASE 67. *Six months hæmaturia, growth, calculi, blood and lymph invasion.*—M.S., æt. 53 (m.). Admitted under Mr. Jacobson, September 3rd, 1903, and died September 10th. He has been a heavy drinker, and has had a hernia for the last seven years. Six months ago he dislocated his right shoulder and bruised his right thorax and loin, and a fortnight later he had hæmaturia for three days. Three months ago he began to have attacks of acute pain in both loins, lasting some five hours at a time, and generally occurring at night. Two months ago the pain became localized in the right loin; for the last three weeks this has been worse, a tumour has appeared, and micturition has been frequent and occasionally painful. On admission: He looks as if he had peritonitis. His abdomen is tense and tender, especially in the right loin, where an ill-defined fluctuating dull tumour is present. Liver enlarged. Pulse 130. Temperature 100·4°. Respiration 48. Urine 1015, acid; a trace of albumen. Leucocytes 32,500. September 4th: A transverse incision was made in the right loin behind and pus let out. He developed bronchitis and died. Post Mortem: 1½ pints of fluid in each side of the chest. Lungs; bronchiectasis, œdema, nodules of growth. Liver contained growth. Right kidney adherent all round and converted into a shell of renal tissue enclosing large masses of growth, pus, and four or five big branched calculi. Growth was present in the appendix, the pancreas was enlarged and congested, the lumbar and mesenteric glands enlarged and hard. Other viscera healthy.

Surg. Rep. 312.

CASE 68. *Surcoma of adrenal and kidney in a child, no metastases.*—E. J., æt. 2 years and 7 months (f.). Admitted under Sir Cooper Perry, May 26th, 1903. Admitted for swelling of the abdomen. Five weeks ago she fell downstairs, and a week later a swelling was noticed in the right side of the abdomen, which has been steadily increasing in size. On May 26th she was sick, and in a good deal of pain. She was brought up to the hospital, and at once admitted. The legs were found to be slightly œdematous and drawn up towards the abdomen, which was much distended, especially on the right side. The superficial veins were dilated, and a hard tumour could be felt extending from 1½ inch above Poupart's ligament upwards to the right of the umbilicus and outwards into the flank, becoming continuous with the liver. There was dulness all over the tumour. No specimen of urine was obtained. The child became collapsed, and died within twenty hours of admission. Post Mortem: No pleurisy; lungs healthy, base of the right one compressed and airless from the abdominal tumour pushing up the diaphragm. Stomach dis-

placed to left, cæcum, appendix, and lower part of ascending colon pushed forward by a large tumour. No ascites. Abdomen distended by a large mass in the right half. No growth in the liver, but the tumour had gradually pushed it over to the left side and at the same time become firmly attached to it, so that it appeared as if the tumour was actually hepatic. On making a vertical section of the mass it was found to be composed of two parts, a lower spherical mass in the right kidney, capped by another much larger mass, which seemed to have originated in the right adrenal, of which no trace could be found. Both tumours were soft and very friable, and in appearance and consistence resembled hæmorrhagic brain substance. Renal vessels not thrombosed. Some normal kidney tissue could be seen forming a kind of capsule to the lowest parts of the tumour. The growth was a small round-celled sarcoma. *Insp.* 1903, 192.

CASE 69. *Adrenal rest tumour, hæmaturia, no metastases.*—J. K., æt. 68 (m.). Admitted under Mr. Golding Bird, June 5th, 1903, and died June 11th. Admitted for retention and hæmaturia. Family history good, personal history: repeated gonorrhœa, syphilis, cystitis, stricture. He has been passing a catheter at intervals. On June 4th he had retention, and a quantity of bloody urine was then drawn off with a catheter, which was tied in, but had to be removed as it soon became clogged with blood. On admission, bladder distended, and renal pain, worse left side. Catheter used and bloody urine drawn off. For the next four days he continued to have hæmaturia, and to be dependent on a catheter. On the 9th the bladder was washed out with hazeline; much blood. Temperature 104°. On the 10th, A.C.E. was given, and a suprapubic cystotomy performed. Blood clot was extracted, and a small prostatic adenoma (thought at the time to be growth). The bladder was washed out with hot water and hazeline, and a tube fixed in. The next day he died. Post Mortem: Old tubercle both apices, pleural adhesions over the posterior and diaphragmatic surfaces of the right lung. Lungs œdematous behind, emphysematous in front, in the left lower lobe were several patches of early septic bronchopneumonia. Pigmentary degeneration of myocardium. Adrenals normal. Kidneys 213 gr, left 130. There was a spherical nodule of growth 4 cm. in diameter, which projected from the upper inner posterior surface of the left kidney. The capsule could be stripped from it, but it was continuous with the kidney capsule below, and could not be separated from it, and it opened into the pelvis, which was considerably dilated and deeply congested. From the appearance it seemed to have bled freely into the pelvis. The growth was soft, hæmorrhagic, and streaked with yellowish areas. From its position and appearance it looked like a neoplasm originating in an adrenal rest. Bladder full of blood clots, some ulceration, a few small polypoid masses at the beginning of the urethra, otherwise nothing abnormal. Mucous membrane deeply stained. Prostate enlarged, no carcinoma. Several phleboliths in the prostatic plexus. Microscopically, the primary growth was a carcinoma of the suprarenal, (probably carcinoma originating in a suprarenal rest was meant, for the adrenals are noted as being normal.) *Insp.* 1903, 213. *Surg. Rep.* 292.

CASE 70. *Adrenal rest growth. Tumour 2 years. Nephrectomy. Recovery.*—G. H., æt. 59 (m.). Admitted under Mr. Golding Bird, November 4th

1904. Family and personal history good. A swelling had been noticed in the left side for two years. A year ago, after an attack of influenza, it began to cause discomfort. There was no pain—only a heavy feeling. There was never any hæmaturia. On admission, the patient's general appearance was healthy. His abdomen bulged on both sides, more markedly on the left. On palpation the right side of the abdomen was normal, and the liver not enlarged. On the left side a large mass could be felt, situated chiefly in the left lumbar region, but extending up into the left hypochondrium and forwards into the umbilical region. This was hard, did not fluctuate, had an ill-defined shape, and moved on respiration. By placing the hand behind the left loin it could be moved downwards and forwards. It was slightly tender. The mass felt uniform all over, except at a point immediately above the anterior superior iliac spine, where a movable tube could be felt, apparently continuous with the sigmoid. In the course of examination, gas could be felt to pass through this. A knob could be felt at the anterior inferior angle of the mass, but no notch could be found. The costal angle was much increased. The bladder was not enlarged, and nothing abnormal was felt per rectum. Urine 1022, acid. No abnormal constituents. A blood count showed nothing abnormal. Fluid was drawn off by an exploring syringe, and on examination showed ordinary blood with a few short chains of cocci. Nothing grew from these in six days at the temperature of the body. Operation: A.C.E. was administered, and the breathing failed. The patient was restored by artificial respiration, and an exploratory incision was made, 4 inches long, vertical, just above the left anterior superior iliac spine. The descending colon was found pushed forwards by an extra-peritoneal tumour which bulged up between the two layers of the descending meso-colon, and reached forwards nearly to the aorta. The pancreas and spleen felt normal. As the tumour was clearly renal, this incision was closed, and an oblique one made for 7 inches from the tip of the last rib to the thickest part of the iliac crest. The quadratus was not divided. The tumour was found covered with a tough capsule. This was divided, and the mass separated out with great difficulty by the hand. During this proceeding the thin inner capsule was torn. The separation of the mass from its outer capsule was quickly performed, and the tumour drawn down and pushed out of the lumbar opening. A large pedicle could be seen, consisting of the renal vessels and ureter; this was secured by a Staffordshire knot of thick silk, divided, and the whole mass freed. Owing to the patient's condition, it was found necessary at this stage to infuse 35 oz. into the axilla. The wound was then washed out with mercury perchloride lotion, and two large drains fixed in. The patient's condition was very bad immediately after the operation, but improved rapidly in the course of the next few days, and is now satisfactory. The tumour will be found figured and described at the beginning of the paper.

A CASE OF SEVERE HÆMOCYTOLYSIS.

BY G. T. WRENCH, M.B., AND J. H. BRYANT, M.D.

IN the following case of a girl, æt. 10, hæmocytolysis was present in such a marked degree, and the blood exhibited such extraordinary changes, that we have deemed it worthy of record. For a few days a fatal result appeared to be inevitable, but recovery took place eventually at the end of three weeks, with complete restoration of the blood to normal.

Florence C., æt. 10, was admitted into Miriam ward on April 19th, 1902, under the care of Dr. Bryant, for profound anæmia. Three years ago she had an attack of measles, otherwise she had always been strong and healthy. Her parents considered her to be a strong child, and stated she rarely suffered from any ailments.

On April 11th, 1902, *i.e.*, eight days before admission, she was playing in the yard when she slipped and fell, striking her left side in the region of the spleen, against the shaft of a cart. She went indoors and complained to her mother of the injury, but soon went out again and resumed her game. She appeared to be quite well for the next two days.

On Sunday, April 14th, she complained of feeling ill; loss of appetite and headache. Her parents thought she had a bilious attack and took little notice until April 16th, when they observed that she had changed colour, her face being pale and of a somewhat yellowish tint. The pallor increased up to the time of her admission on April 19th. During this time she was able to take nothing but milk, or milk and egg. She was sick after food,

usually about three times a day, was very thirsty and complained of her tongue being rough and dry. There was no evidence of hæmorrhage and she had no rash. She was constipated for three days. She had neither headache nor mental symptoms.

Her parents are strong and healthy. She has six brothers and sisters, all of whom are living and well. On Friday, April 19th, she was admitted into Miriam ward.

Condition on admission.—Temperature 98°, pulse 96, respirations 24. She was a well-nourished child but markedly anæmic. The skin, however, had a curious yellowish-brown tint more like that of Addison's disease than of simple anæmia. She was very restless and was continually yawning. She had a wild look about the eyes although she was not delirious.

Circulatory system.—The impulse was seen and felt in the fifth left intercostal space, just beyond the nipple line. There was no thrill. The superficial cardiac dulness commenced above in the third left space and externally extended just beyond the nipple line. At the apex there was a systolic murmur which could be heard all over the front of the chest, but which was best marked in the pulmonary area. It was also traceable into the carotids and along the abdominal aorta and external iliac arteries. During a full inspiration the heart-beat was much slowed. There was a doubtful bruit de diable just above the clavicle but it was difficult to dissociate it from the systolic bruit.

Abdomen.—The abdomen was not distended. It was supple and there was no tenderness. The edge of the liver could be felt just below the costal margin in the right nipple line.

The spleen was enlarged. It could be felt half an inch anterior to the left nipple line, and below, it extended to within a finger's breadth of the level of the umbilicus. It was not tender. The edge was regular, sharp, and well defined.

Skin.—No rash present.

Respiratory and nervous systems.—There were no abnormal signs.

The urine was acid and contained neither blood, sugar, pus nor albumen.

The vomit contained no blood and no free hydrochloric acid.

The *fæces* were carefully examined for parasites and ova but none were detected.

Blood.—(In describing the different kinds of blood-cells, the nomenclature of Ehrlich, as given by Cabot (3rd edition) was used and all differential counts were made from specimens stained by Ehrlich's triacid stain. The blood-counts were made with the Thoma-Zeiss hæmocytometer and the hæmoglobin was estimated by Marie's hæmoglobinometer, and once by the specific gravity method).

April 19th.—

Red blood corpuscles	...	939·300	per cubic millimetre.
Hæmoglobin	...	27	per cent.
Nucleated red corpuscles	...	27·600	per cubic millimetre.
White	...	69·000	"
Polymorphonuclear	...	66·4	per cent.
Small lymphocytes	...	22·2	"
Large lymphocytes	...	2·4	"
Myelocytes	...	1·8	"
Eosinophiles	...	2·0	"
Eosinophile myelocytes	...	0·2	"
Transitional	...	5·0	"

A microscopical examination of the films showed the majority of the red corpuscles to be undersized. A large number of red cells were nucleated. The nuclei were bipartite, tripartite or irregular. Some of the bipartite cells were dividing in equal proportion and with division of the protoplasm, suggested cell division. Some of the nuclei were deeply stained, probably old corpuscles, others, especially those of the large erythroblasts, were pale blue. Many of the red corpuscles showed well-marked polychromatophilia. There was no poikilocytosis, the corpuscles retaining their normal contour, nor were there fragments of broken corpuscles. The white corpuscles, although greatly increased in number, did not display any disproportion in their relative percentage, except for the presence of a few myelocytes.

She was given an ounce of peptonized milk every hour and large nutrient enemata every six hours.* 0·6 c.c. of cacodylate of soda solution was injected into the axilla at 10 p.m.

* A sterile solution was used which contained 0·05 gm. *i.e.* $\frac{1}{20}$ grain of Cacodylic Acid in 1 c.c. (17 minims).

She was very restless during the night and made several attempts to get out of bed, and was once found sitting on a chair by her bed.

April 20th. She was in much the same condition as on admission. She was rational with her own relations, but with nobody else. The bowels were opened, but both urine and fæces were passed unconsciously. The motion was very dark, but contained no blood. There was no epistaxis, hæmatemesis, melæna, hæmaturia nor purpura. She did not complain of pain. The spleen was the same size as on the 19th. Her grandmother, who had watched her from the beginning of her illness, considered her colour better to-day. The highest recorded temperature was 99·8° at 10 a.m. At 6 p.m. the pulse was 160, and the lowest rate was 136 at 10 a.m. Urine: 1022, pinkish deposit of urates, urea 2·05 per cent., no albumen, sugar, bile, pigment or indican; a little urobilin was detected. At 9 p.m., 0·9 c.c. of cacodylate of soda solution was injected into the axilla.

The eyes were examined and a few retinal hæmorrhages were seen.

The blood-count showed the following:—

Red blood corpuscles	1,164,500
White blood corpuscles	105,500

The differential count was not markedly different to that made on the 19th, except for the smaller percentage of polymorphonuclear cells (53·8 per cent.), the larger percentage of small lymphocytes (28·8 per cent.), and the appearance of new cell-like bodies which were not present in the films made and examined on the 19th.

They did not conform to any type of leucocyte described by Cabot, Ewing, or Von Limbeck. They have been placed in the list of the differential counts amongst the leucocytes and named "vacuolated bodies." In appearance these "vacuolated bodies" presented fairly definite masses of protoplasm which took on basic stains either lightly or darkly, but as a rule to a tint that was less deep than the nuclei of the concomitant leucocytes. This stained protoplasm was much vacuolated, and was bound

together by a kind of fibrillar network. The margins of the vacuoles were either closely defined or merged into the blue-stained protoplasm. These bodies varied greatly in size but the average was considerably above the measurement of the leucocytes. Some only measured 9μ by 10μ , but many as much as 16μ by 14μ or even 12μ by 28μ .

At first it was thought they might be basophiles, but their staining reactions with Jenner's stain and methylene blue did not correspond with this kind of cell. True basophiles were seen only on one day (April 24th). The shape of the vacuolated bodies was often irregular with protrusions comparable to the pseudopodia of *amœba*. A few specimens of fresh blood were examined, but nothing of a parasitic nature was noticed. No special search with a warm stage was made for parasites, as this interpretation of the bodies was not thought of at the time. They had no definite nucleus, and no granules.

With Jenner's stain they presented a similar uniformly stained vacuolated mass. With eosin and methylene blue they presented the same appearance as with Ehrlich's triacid or Jenner's stain. Sometimes similarly stained uniform, but unvacuolated, protoplasm was seen with fine oxyphile granules near them and occasionally these fine granules were observed apart from any protoplasm, as if lying loose in the blood-stream or separated from their cell in the process of making the film.

April 21st. She was in much the same condition, but the spleen was a little smaller. The tendency to delirium was still present. The eyes were examined and hæmorrhages into both retinæ were found. She appeared to have a slightly better colour. She was still troubled with vomiting. The highest recorded temperature was 99.4° at 2 p.m., and the lowest 97.8° at 6 p.m. A few uric acid crystals were found in the urine.

She was given some bread and milk, but vomited it soon afterwards. Plasmon 3j. and Extract of Malt 3ii. was ordered for her, to be taken three times a day.

22nd. She was improving in every respect, being more sensible and of a better colour, and the spleen was appreciably

smaller; 13 minims of cacodylate of soda solution were injected into the axilla.

23rd. She was less anæmic in appearance and much more sensible. The spleen was smaller. The temperature was 99°; 20 minims of cacodylate of soda solution ($\frac{1}{7}$ grs.) were injected subcutaneously at 11.45 a.m.

24th. A further improvement in her colour and also in her manner was noticeable. The spleen was a little smaller. Urine acid, no albumen, urea 2.4 per cent.; no uric acid crystals; Indigo red present but no indican blue: no urobilin. 14 minims of cacodylate of soda solution ($\frac{1}{8}$ gr.), were injected into the axilla at 11.45 a.m.

25th. She appeared to be steadily improving in every respect; 10 minims of cacodylate of soda solution were injected.

26th. The spleen was smaller. Urine: 1025; acid, urobilin present. 15 minims of cacodylate of soda injected.

27th. She felt and looked comparatively well. She was now able to take (minced) fish for dinner and bread and milk for breakfast; 15 minims of cacodylate of soda solution injected. Urine 1025; a good deal of urobilin was present.

28th. The administration of one-fifth grain of cacodylate of soda in the form of a pill three times a day was now commenced.

29th. She looked quite well. She had no pain and was able to take full diet without any discomfort.

May 1st. The mucous membranes were still a little pale. Systolic murmurs were audible in the pulmonary and mitral areas. The cardiac impulse was in the fifth left space about three-quarters of an inch outside the nipple line. The spleen was just palpable below the left costal margin.

3rd. The spleen was not palpable.

9th. She was discharged, having been up and about the ward for several days. Her colour had greatly improved. She had a distinct red tint about the cheeks, but elsewhere the skin was sallow. She was no longer sick and the bowels acted regularly every morning. No hæmic murmurs could be detected. Neither the spleen nor the liver could be felt beneath the costal margin. The retina presented a normal appearance.

One of the most interesting points to be determined in this case was the cause of the remarkable changes in the blood. The parents attributed her condition to the injury she had received in the region of the spleen and the enlargement of that organ, which was discovered on her admission with its subsequent diminution in size as she recovered, seemed to corroborate this view and to point to some injury or to some derangement in the function of the spleen as the most likely cause of the anæmia.

What pathological changes would account for such an enormous decrease in the number of red blood corpuscles (939,333 per c.mm. *i.e.* to about 18 per cent. of normal) the relative large amount of hæmoglobin (27 per cent.) giving a colour index of 1·5, the leucocytosis (69,000 per c.mm.) and the extraordinary number of nucleated red corpuscles (27,600 per c.mm.)?

In all respects, with the exception of leucocytosis, the changes resembled those which are usually found in advanced cases of pernicious anæmia. In all marked examples of this disease the leucocytes are diminished in number, frequently below 4,000 per c.mm. and even to 2,000 or 1,500. Leucocytosis, when it does occur in the course of pernicious anæmia in adults is invariably due to some such complication as hæmorrhage or suppuration, but in children it takes much less to induce leucocytosis. In this case no complication was discovered to account for it. Pernicious anæmia is rare at such an age as ten, and further, the onset is much less acute and recovery is much more protracted; additional reasons which help to exclude a diagnosis of this disease.

The extreme pallor, the restlessness, and the constant yawning suggested the possibility of a severe hæmorrhage. There was no history of epistaxis, hæmatemesis, hæmoptysis, nor of any other form of hæmorrhage, nor did a careful physical examination of the chest and abdomen yield any signs of an internal hæmorrhage.

The injury to the left side and the enlargement of the spleen made us consider whether that organ had been ruptured and bleeding had occurred into the peritoneal cavity.

A simple hæmorrhage, however, would hardly account for such marked changes in the blood. As a result of the loss of a

considerable quantity of blood there is a diminution in the number of red blood corpuscles, and the hæmoglobin is much reduced, so that the colour index is usually below one. The presence of such a large number of nucleated red corpuscles was compatible with a severe hæmorrhage, for after the loss of a considerable quantity of blood it is usual to find a large number of normoblasts and microblasts in the blood. Ewing writes, "Small and repeated hæmorrhages, on the other hand, have led to some of the most severe forms of anæmia ever recorded in which the morphological changes of pernicious anæmia are pronounced, but the prevailing feature of the blood is the loss of hæmoglobin." It is usual to get a leucocytosis after hæmorrhage, but the number of leucocytes per cubic millimetre do not as a rule exceed 40,000. A differential count shows a relative lymphocytosis. There are no grounds for supposing that in this case there had been repeated small hæmorrhages. If the anæmia was due to hæmorrhage it must have been connected with the injury to the side and to bleeding into or from the spleen.

The enlargement of the spleen suggests the possibility of some disorder or derangement of the function of that organ as a result of the injury.

In two cases of splenectomy (of the healthy spleen) mentioned by Dr. Rolleston (Clifford Allbutt's *System of Medicine*, vol. iv.), the following group of symptoms occurred:—

1. Progressive loss of strength and weight and of emaciation.
2. Extreme anæmia.
3. A daily rise of temperature 1° to 3° Fahrenheit.
4. Increased frequency of the pulse.
5. Fainting attacks, with increased pallor of the surface.
6. Headache, drowsiness and great thirst.
7. Severe gripping pains in the abdomen and pain in the legs and arms. In one case tenderness along the tibia, which was thought to indicate compensatory changes in the red marrow of the bones.
8. Enlargement of the external lymphatic glands, which remained permanently increased in size.

9. Blood changes, which consisted of a diminution in the number of the red blood corpuscles and an increase in the number of leucocytes.

In this case, with the exception of 7 and 8, all these symptoms were prominent and striking, especially the diminution in the number of red blood corpuscles and the increase in the number of leucocytes.

Could the injury, which she had received in the region of the spleen, in some obscure manner, have put that organ temporarily out of action, as when splenectomy removes the splenic function from the living subjects?

This explanation, however, was by no means satisfactory. In the first place, we were not able to find any previous record of such severe hæmocytolysis and illness following an injury in the splenic area, and in the second place, the injury was slight, for there was no external evidence of it in the form of bruising, and the child, as has already been stated, was quickly enabled to return to her play.

Casting around for some other cause we naturally thought, in the absence of any evidence of hæmorrhage, of the possibility of her condition being due to the influence of some poison. Cabot (Clinical Examination of the Blood, 3rd edition) quotes a case reported by Brandenburg of acute anæmia with marked leucocytosis as the result of poisoning by chlorate of potash and another case, reported by Ehrlich and Lindenthal of a similar condition due to poisoning by nitrobenzol.

This last mentioned case is also quoted by Ewing (Clinical Pathology of the Blood, 1st edition, 315). The blood became chocolate-coloured ten hours after the initial symptoms and methæmoglobin was visible by spectral analysis until the eighth day. On the fifth day the red cells were reduced to 2,275,000 and before death fell to 300,000 per cubic millimetre. Poikilocytosis was noted on the third day. Nucleated red corpuscles were first seen on the third day, and on the ninth day 24,700 to the cubic millimetre were counted. On the ninth day the leucocytes rose to 61,000. The hæmoglobin fell to 40 per cent., so that with 900,000 red blood corpuscles to the cubic millimetre

and this percentage of hæmoglobin the colour index was remarkably high. "The morphological characters of the blood described by Ehrlich and Lindenthal probably represent an extreme degree of the effects upon the blood of the entire group of anilin poisons."

Some such poison, we thought, might have been the cause of the hæmocytolysis in our case but all enquiries and efforts at discovering such a cause proved to be unavailing until six weeks after the patient's discharge, when our suspicions received confirmation in an unexpected manner. The quarterly gas bill was sent to the father of our patient and found to be considerably more than usual, in fact was nearly double the amount of the corresponding quarter for the previous year. Recalling our persistent questioning as to the possibility of his daughter having taken some poisonous substance, he looked for a leak in the gas-pipe of her bedroom and discovered an escape of gas which was situated under the floor immediately over which her bed was placed. We feel justified therefore in attributing the illness of our patient to the subacute toxic effects of coal gas.

We did not notice any marked difference in the general naked-eye appearance of the blood; it certainly was not a bright cherry red nor was it chocolate coloured. We did not obtain a history suggesting hæmoglobinuria, nor after admission did we find any evidence of such a condition. Although we have not been able to find a similar case of anæmia which has been attributed to chronic coal gas poisoning, we are of opinion that this form of toxæmia is the most satisfactory explanation of this interesting and remarkable case.

The rapid recovery was also of very great interest, but whether it was due to the treatment with cacodylate of sodium or to the change of environment, and so the removal of the apparent cause, we prefer to leave an open question.

She was examined two months after discharge. She was quite healthy and the blood-count was normal.

A CASE OF SEVERE HÆMOCYTOLYSIS.

Day of Disease.	Mar. 19, 1902	April 20, 1902	April 21, 1902	April 22, 1902	April 24, 1902	April 26, 1902	April 30, 1902	April 2, 1902	May 5, 1902	Remarks.
Total number of reds per c.mm.	989,333	1,164,477	1,299,417	993,750	1,299,896	1,625,000	2,364,327	3,575,000	5,280,000	
Total number of whites.	69,000	105,893	82,458	81,250	40,105	25,000	13,183	10,000	11,200	*Taken actually on the 25th by the spec. grav.
Hæmoglobin ...	27 %	—	—	32 %	—	40 %*	55 %	65 %†	75 %	†This was taken on April 5th. It is inserted here for the sake of comparison.
Colour index ...	1.5	—	—	1.6	—	1.3*	1.3	0.93†	0.8	
Proportion of white to red	1:17	1:11	1:16	1:13	1:305	1:65	1:172	1:350	1:471	
Proportion of nucleated red to white	1:2	1:4	1:2	1:12	1:106	1:285	—	—	—	
Proportion of nucleated red to red	1:34	1:44	1:33	1:15	1:31	1:3000	—	—	—	
<i>Reds not showing mitosis—Differential count.</i>	500 counted	500 counted	500	500	500	200 a bad slide	300	300	300	
Polymorpho-nuclear mu-trophils	66.4 %	53.8 %	77.0 %	56 %	57.8 %	61 %	62 %	63 %	57 %†	‡These include loose and torn cells with fine oxyphile granules and pale blue nuclei.
Small lymphocytes ...	22.2 "	28.8 "	20.8 "	29.4 "	27.1 "	30 "	30.3 "	29.3 "	36 "	
Large lymphocytes ...	2.4 "	3.2 "	1.8 "	3.2 "	4.6 "	5 "	6 "	6 "	3.8 "	
Eosinophiles ...	2 "	6.6 "	0.6 "	0.2 "	1.9 "	1.0 "	0.6 "	1.7 "	1.1 "	
Myelocytes ...	1.8 "	2.6 "	1.8 "	1.8 "	1.8 "	1.2 "	—	—	—	Transitional between polymorph. and lymphocytes were small mononuclear (nomad) neutrophiles.
Eosinophiles myelocytes	0.2 "	—	—	—	—	—	—	—	—	
Basophiles ...	—	—	—	—	0.1 "	—	—	—	—	

A CASE OF SEVERE HEMOCYTOLYSIS—continued.

Day of Disease.	Mar. 19, 1902	April 20, 1902	April 21, 1902	April 22, 1902	April 24, 1902	April 26, 1902	April 30, 1902	April 2, 1902	May 15, 1902	Remarks.
Vacuolated bodies	— %	5 %	1.4 o/o	4.6 %	3.4 %	0.6 %	0.6 %	—	1.1 %	¶These were definite masses of protoplasm, much vacuolated, bound together by a sort of fibrillar network. They varied greatly in size, measuring from 12 μ up to 30 μ across.
Transitional between polymorph. & myelocyte	1.4 "	1.4 "	2 "	1.6 "	—	—	0.3 "	—	—	
Transitional between polymorph. & lymphocyte	1.6 "	0.8 "	0.4 "	0.6 "	0.5 "	—	—	—	—	
Transitional between small and large lymphocytes	2.0 "	3.8 "	1.2 "	2.6 "	2.8 "	0.6 "	—	—	1 "	
Sizes of red cells.	Of each measured				50 measured					
Nucleated red	100									
5.4 μ	22	—	—	—	24	—	—	—	no nucleated	Taking 5-9 as the normal limit of a red blood cell, there was at first a general undersize, no megalo-cytes, and but few megalo-blasts. Many nucleated red had light blue nuclei young corpuscles.
7.2 μ	46	—	—	—	23	—	no nucleated	—	reds	
9.0 μ	27	—	—	—	3	—	reds	—	100	
10.8 μ	4	—	—	—	—	none measured	100 measured	none measured	measured	
12.6 μ	1 100	while counting whites	while counting whites	none measured	100 measured					
Non-nucleated red	29	1 nucleated	3 largest nucleated	—	19	—	—	—	—	
3.6 μ		red measured	red measured	—	32	—	23	—	—	
5.4 μ	59	red measured	14.5 μ	—		—				
		12.5 μ × 11 μ other-wise	11 μ × 11 μ	—	42	—	56	—	—	
7.2 μ	9	no megalo-blasts	11 μ × 9 μ	—	7	—	21	—	—	
9.0 μ	3 100									

Polychromatophilia present at first and slightly on 30th. No poikilocytosis.

A COLLECTION OF CASES IN WHICH THE OPERATION OF EXCISION OF THE HIP-JOINT HAS BEEN PERFORMED FOR DISEASE OF THE JOINT.

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SURGICAL REGISTRAR.

THE object of the appended collection of cases is, primarily, to elicit the history of those patients in whom excision of the hip-joint has been performed for disease of that joint, and in whom the operation has succeeded, either partially or completely, in arresting the disease.

But, in order that the collection of cases may be made more just, fuller, and more interesting, there have been added those cases in which disease of the hip-joint continued, and eventually resulted in amputation, through the upper part of the thigh, or in death, a previous excision of the hip-joint having been performed.

It is advisable to point out in this place that the term "disease of the hip-joint" indicates tuberculous disease, but two cases have been included in the collection which were probably not tuberculous (cases 10 and 30).

Forty cases in all have been traced since the operation of excision of the hip-joint was performed. Letters were addressed to many more, but in answer to some, no replies were received;

and, it may fairly be assumed that some of the cases were prevented from answering letters, or attending personally at the hospital, by the intervention of death. Of these forty cases which have been traced, definite information of their history after excision has been secured in all but three cases, concerning which, however, some information has been obtained.

These forty cases, with four exceptions, have been under the care of surgeons at Guy's Hospital during the years 1896 to 1903. Two patients were operated upon by Sir Henry Howse, and Mr. Symonds respectively, before 1896. Two patients were operated upon at other hospitals.

The interval which has elapsed between the excision of the hip and the examination of the patient by the writer, or the records of actual events in the surgical reports of Guy's Hospital, varies from nineteen years to six months. In two cases, more than ten years have elapsed between the operation of excision, and the writer's examination, or the actual records of failure or success; in fourteen cases more than five years; in four cases more than three years; in twelve cases more than one year; whilst in five cases less than a year has intervened between the operation of excision of the hip and examination.

Three cases have not been included amongst these figures, owing to death immediately following excision of the hip in two cases, and incompleteness of history in the third case.

The results may be considered under three heads; first, the cure of the tuberculous process; secondly, the deformity produced by the operation; thirdly, the utility of the limb after the operation, and the ability of the patient to enter upon the various duties of life after excision of the hip has been performed. The cure of the tuberculous disease may first be considered.

Three results may occur after excision of the hip. First, in some cases excision has had eventually to be followed by amputation, through the hip-joint, or a fatal termination has occurred at some time after the operation. These cases have been reckoned by the writer as failures. Secondly, those cases in which there is no sign of continuation of disease, as evidenced

by abscess or sinus-formation, have been reckoned as successes. Thirdly, those cases which still show sinus-formation, or evidence of abscess-formation, have been reckoned as partial failures. There is certainly a difficulty in accurately classifying these partial failures, for whilst some are undoubtedly progressing towards recovery, others are as surely progressing towards amputation, or perhaps even early death.

In six cases, out of the total number collected (15 per cent.) death has occurred since the operation. Two deaths resulted from shock immediately following surgical interference (cases 24 and 33). Two deaths have occurred from progressive tuberculous or lardaceous disease (cases 21 and 35); whilst two patients have died outside the hospital (cases 2 and 25). As the last two cases were discharged in a considerably worse condition than when they were admitted, it may be justly considered that death occurred from complications or sequelæ associated with the hip trouble. In two of these cases death took place in spite of amputation through the hip-joint

Eight other cases are known to have had amputation performed at the hip-joint after excision of the joint. Thus excision of the hip-joint resulted in failure in fourteen cases (35 per cent.).

The failures, represented either by death or amputation through the hip-joint, excluding the deaths actually due to operation and excluding an unusual case of nineteen years' interval, occurred on an average three years after excision of the hip-joint

There are in the present series of cases eleven partial failures, that is to say, that in 27·5 per cent. of the cases there are still sinuses present. In fifteen cases, or in 37·5 per cent. the results of excision of the hip-joint have been successful.

A more detailed analysis of the cases may now be made. There are, in this collection of cases, fourteen females and twenty-six males. Whilst in each sex there are eight successes, there are five failures amongst the females against nine failures amongst the males, and one partial failure amongst the females against nine partial failures amongst the males. These figures seem to show that the males have the same power to resist the progress of disease after excision as the females, but, owing no doubt to

greater activity in life, have less power to resist the continuation of disease.

For the purposes of comparison with other forms of treatment of hip disease the following facts may be stated. The cases of excision of the hip may be divided into three groups. The first group comprises those cases in which excision of the hip-joint has been performed before the formation of an abscess or sinus. In this group there are seven cases with three successes, three failures and one partial failure. The second group comprises those cases in which an excision was performed, when abscess-formation was present, but no sinuses. In this group there are twenty cases, made up by seven successes, seven failures, and six partial failures.

The third group comprises those cases in which sinus-formation had occurred when excision was performed. In this group there were nine cases with three successes, two failures and four partial failures. Four cases in which the history was imperfect have not been included under these headings. The mortality in the first group was 14 per cent., in the second 18 per cent., in the third 22 per cent.

The failures, that is those cases in which death occurred after, or amputation followed, excision, amounted in the first group to 43 per cent., in the second to 35 per cent., and in the third to 22 per cent. Thus, as regards the failure or otherwise of excision of the hip, it may be concluded that excision is more favourable when an abscess has formed than when it has not, since failures are more common with excision performed before, rather than after, an abscess has formed.

As regards the age of the patient, there were sixteen cases over the age of ten whose hips were excised; of these there were eight successes, or 50 per cent. of the total number. In twenty-four cases operated upon under the age of ten, there were eight successes, representing 33 per cent. of the total number. In none of the cases actually examined by the writer, twenty-three in number, was there any evidence of advancing lardaceous or tubercular disease, but in eleven cases there were sinuses present near the site of operation, indicating continuation of the disease. Many of these sinuses appeared superficial, and a

single scraping might have been expected to effect a cure, but if the vagaries of the tuberculous process be considered, even these cases cannot fairly be considered as cures. Sinuses, however, do not appear to prevent patients from enjoying life or earning money. Thus, one case (29) whose hip was excised by Mr. Lucas was delighted to come up from Worthing and to spend a few days in town. He was thoroughly happy, although about every three months a sinus appeared, which quickly healed up. Another patient (38) performed his duties as night watchman although sinuses are still present (May, 1904).

Deformity.—The next point to be considered is that of the deformity produced by the operation of excision of the hip. The deformity is usually well marked, and is chiefly due to the shortening of the limb on the side of the excised joint.

In every case examined with a view to discovering the amount of shortening, the writer found that the great trochanter is displaced directly upwards on to the dorsum ilii, the tip of the great trochanter being generally nearly as high as the level of the anterior superior spine on the same side. In the great majority of cases this displacement of the great trochanter upwards is responsible for all the shortening; but in six cases there was marked shortening below the knee, amounting in each case to at least an inch (cases 12, 14, 23, 26, 28 and 30).

It is not easy to obtain an exact history of the forms of splints used, or length of time for which they were applied after excision, but the displacement upwards of the trochanter occurred irrespective of the application of splints; thus, in one case splints were still being used seven years after the operation of excision, and in this case the displacement upwards was just as marked as in those cases in which splints had been discarded shortly after operation.

Moreover, in those cases where special efforts were made by the surgeon, either by the introduction of wire joining the upper part of the femur to the acetabulum, or by special care exercised over the application of splints and position of the limb, the displacement upwards of the trochanter was just as

marked eventually as in those cases where no such special precautions were taken (*vide* cases 1 and 15).

In some of the cases of excision of the hip seen at the Out-patient department, lordosis of the spine indicating flexion of the artificial joint is marked, but in the writer's series of cases, out of twenty-two observed, only seven showed lordosis.

The obvious disadvantage of flexion of the artificial joint is the great increase in apparent deformity which it causes, and the enormous boot which must be worn by the patient; in one case a boot with a sole nine inches thick was being worn by the patient, and in this case there was only three and a half inches actual shortening. In no case was there scoliosis of a permanent nature.

In conjunction with the deformity, the question of wasting of the whole limb may be considered.

In all the cases seen, the wasting was very apparent on the same side as the excision; and in these cases where the results have otherwise been successful, massage might be tried with advantage, as not only might it strengthen the lower limb, but it might also prevent the shortness of the tibia which occurs in some cases, and further might improve movements in the knee and ankle; and thus further compensation than that afforded by the pelvis and spine might be afforded for the loss of the natural hip-joint.

Utility of the joint.—The utility of the new joint after excision now demands attention. In all the cases seen by the writer, the utility of the new joint was good, except in one case (28), in which an excision of the hip has been performed, and in which, for all the use the patient was making of the limb, amputation of the lower limb at the hip-joint might just as well have been performed as excision.

In all the cases, except three, the whole weight of the body could be borne upon the artificial joint between the dorsum ilii and the femur. In no case did the great trochanter rise in the slightest degree when this test was carried out.

Two of the most intelligent patients told the writer that they could do everything in the way of movements, except bend down

in such a way as would enable them to fasten their shoe on the side of the excised joint.

An interesting question which may now be raised is that of mobility in the new joint. The writer was led to believe, before he made enquiries for himself, that flail joints were fairly common after excision of the hip. They are, according to the present collection of cases, rare. In this series only one case is found. In seven cases there was free mobility of the new joint, and in three cases there was partial mobility. In thirteen cases the joint was fixed. The mobility of the joint seems, however, to influence the formation of sinuses, for in eleven cases of movable joint, sinuses were present in six, and in thirteen cases of fixed joint three only had sinus-formation.

These figures are not perhaps of much value if considered by themselves, but when taken in conjunction with the experience of the relative value of excision or erasion of the knee-joint, they seem to indicate that disease is more likely to recrudesce with a movable than with a fixed joint.

In the hip-joint, mobility after excision may be exceedingly useful to the patient; thus, a child, Alice E. B. (not included in collection of cases), was admitted into the Waterloo Hospital for Children in 1899. Her right hip was excised by Mr. Marmaduke Sheild in the same year. The wound healed up and she was discharged. In 1902, she was re-admitted for left hip disease. Since then she has been using crutches, and wearing a patten on the right boot, so that the right hip has been in constant use for two years. The artificial joint is movable in every direction, and the great trochanter does not rise higher than its fixed position on the dorsum ilii, when the weight of the whole body is thrown upon the artificial joint.

Finally, the ability of patients to attend to the ordinary duties of life after excision of the hip has been performed may be considered.

Seven patients, who had reached the money-earning period of life, were obtaining fair wages and working hard. One was working nine hours a day as a clerk and earning thirty shillings

a week ; another was earning one pound a week ; another was earning eight shillings a week, and was working eleven hours a day ; others, younger, were earning five shillings a week or thereabouts ; whilst another, preferring to live the life of a pitiable invalid, though quite capable of working, received eight shillings a week from clubs. A female patient, whose hip was excised by Mr. Golding-Bird for acute arthritis, following upon a miscarriage, told the writer that she had had after the operation the best labour and confinement she had ever experienced. One of the female patients living in Bermondsey, aged twenty-two, and exceedingly pretty, was still unmarried, and for this condition of single life, apart from private reasons which the writer did not enquire into, the deformity must be held responsible.

The preceding remarks and the conclusions which may fairly be considered to arise therefrom may be now summarised in round numbers. Out of two hundred cases in which the hip-joint has been excised, seventy may be expected to be the subjects of progressive disease, and of these thirty may be expected to die from continuation and spread of the disease. Fifty-five will rank as partial failures, in that in these sinuses will persist. In many of these cases, however, sinuses will not interfere with the wage-earning capacity or happiness of the individual. Seventy-five cases may look forward to a complete recovery. Two cases, however, amongst these will have a flail joint. The majority of cases, whether sinus-formation is present or not, will have useful joints. The best result, however, namely, a useful joint, with free mobility and without any sinus, only falls to the lot of twenty-five people out of two hundred, there being in this series of forty cases only five such excellent results.

SYNOPSIS OF CASES.

These are arranged in three classes :—

1. Those cases in which excision was performed for hip-disease, and in which neither abscess nor sinus-formation had occurred before excision.
 2. Those cases in which excision was performed for hip disease and in which an abscess had formed, but no sinus-formation had taken place before excision.
 3. Those cases in which excision was performed for hip disease and in which an abscess had formed and sinus-formation had taken place before excision.
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Class 1 comprises cases numbered from 1 to 7 inclusive.

7 cases—3 successes ... Cases 1, 3, 5.

3 failures ... Cases 2, 6, 7.

1 partial failure (that is sinuses still existed after excision) Case 4.

The successful cases showed two movable and one fixed joint.

The cases which terminated in failure showed one death and two amputations through the hip-joint. In the cases of failure the joint was movable.

CASE 1.—Alice B., æt. 16. Mr. Golding-Bird, 4, 1898. Twelve months' history of hip disease previous to admission on 27th October, 1897. Excision of hip on 9th December, 1897. No sinuses nor abscesses found. Healing took place by primary union. Efforts made to keep femur in acetabulum, and one month after operation there is a record of one inch shortening. Splints and crutches discarded two months after discharge from hospital. On May 21st, 1904, six and a half years after operation, general and local condition was good. No signs of sinus-formation were present. The excised joint was movable and useful. The great trochanter did not rise at all when the weight of the body was borne upon the excised joint. There was two inches shortening due to slipping up of great trochanter. There was wasting of the thigh on affected side, amounting to two inches, and of leg three-quarters of an inch. This patient works eleven and a half hours a day as button-hole maker, earning eight shillings a week, and although decidedly pretty, is unmarried.

CASE 2.—Fred D., æt. 8. Sir Henry Howse, 130, 1896. Admitted March 26th, 1896. Hip disease in second stage. Excision of hip, May 1st, 1896. No sinuses nor abscesses found. Patient made no progress for some time, and on August 11th, 1896, amputation, through the hip-joint, was performed. Sinuses persisted after amputation, and the patient died on August 10th, 1898, at home. When he was discharged from Guy's Hospital, he shewed signs of phthisis at the apex of the right lung.

CASE 3.—Elizabeth H., æt. 6. Sir Henry Howse, 126, 1901. Six months' history of hip disease. Head of femur excised, 15th March, 1901. Complete healing by June 30th, 1901. Sinus appeared and was scraped in August, 1901. Patient discharged with sinus practically healed, on September 20th, 1901. Examined in May, 1904, three years after operation. Three inches shortening on affected side, made up by slipping up of great trochanter. Great trochanter does not rise when weight of body is borne upon excised joint. Joint fixed, and useful. Wasting of thigh amounts to one and three-quarter inches; wasting of leg to half an inch. Lordosis present, amounting to twenty degrees of flexion of artificial joint. Patient is in good local and general condition.

CASE 4.—Rose S., æt. 6. Mr. Davies Colley, 417, 1897. Nine months' history of hip disease. Excision of head of femur, 15th June, 1897. No abscess nor sinus present. Healing occurred by July 12th, 1897, by primary union. Examined May, 1904, seven years after operation. Three-quarters of an inch shortening, due to slipping up of great trochanter. Great trochanter does not rise when weight of body borne upon excised joint. The joint is movable and useful, and whilst the patient's general condition is very good, there is sinus-formation present.

CASE 5.—Ernest T. H., æt. 22.—Mr. Golding-Bird, 254, 1897. Two years' history of hip disease. Head of femur excised, 10th November, 1896. Healing practically complete when patient was discharged on February 3rd, 1897. Examined in May, 1904, seven years after operation. One and a quarter inches shortening in the affected limb, due to slipping up of the great trochanter. The great trochanter does not rise when the weight of the body is borne upon the excised joint. Joint movable and useful. Wasting of thigh amounts to one inch, and of leg to one and a half inches. There is no spinal curvature; good local condition. Although this patient has a cured tuberculous condition of his left knee and has lupus on his left heel, his general condition is good and he is considered as a success.

CASE 6.—George L., æt. 7. Mr. Jacobson, 213, 1903. One year's history of hip disease. Excision of hip when patient was aged seven in 1896, by Mr. Davies Colley. No mention of abscess or sinus. Sinuses formed after operation. Patient was able to flex his hip through sixty degrees, but other movements were absent. Mr. Jacobson performed amputation through the thigh on 28th July, 1903.

CASE 7.—Arthur H., æt. 20. Sir Henry Howse, 390, 1899. Three years history of hip disease. Excision of hip on the 28th of July, 1898. No abscess nor sinus-formation. Sinuses formed after excision, and amputation through hip-joint, performed on the 18th of July, 1899. Dr. Emil Hardenberg tells me that the condition of the patient is quite good now.

The second group (Nos. 8 to 27), comprises those cases in which excision was performed when an abscess had formed, but when there was no sinus-formation, and comprises twenty cases in which there were seven failures, namely, two deaths and five amputations (cases 11, 19, 20, 21, 24, 25 and 26). There were seven successes, in three of which the joints were movable (cases 8, 18 and 23), and in four of which the joints were fixed (cases 9, 10, 12 and 26). In four cases of partial failure (13, 14, 15 and 17), the joint was in each case fixed :—

CASE 8.—Daniel S., æt. 5. Sir Henry Howse, 1901. One year's history of hip trouble. Abscess had formed and was discovered over great trochanter on October 11th, 1900. Excision of hip on October 18th, 1900. The wound had almost healed on November 23rd. Examined July, 1904, four years after operation. One and a quarter inches shortening, due to displacement upwards of great trochanter. Great trochanter does not rise when the weight of the body is borne upon the excised joint. The hip-joint on the other side is in the second stage of hip disease. The local condition on the side of the excised hip is bad, sinuses being present. The excised joint is movable in all directions, except that abduction and internal rotation are not possible.

CASE 9.—Jessie N., æt. 10. Mr. Lucas, 377, 1897. Two years' history of hip disease. Abscess formed anteriorly; no sinus. Excision of hip performed on the 17th of November, 1896. Healing by primary union was complete by the 3rd December, 1896. A special note is made in the report, that shortly before patient's discharge from hospital there was one inch shortening on the affected side. Examined in May, 1904; seven years after operation. Two and a quarter inches shortening on affected side, due to slipping up of great trochanter. Great trochanter does not rise when the weight of the body is borne upon the excised joint. The joint is fixed and useful. Some lordosis, although not marked, is present. Wasting of thigh amounts to three inches, and of leg to one and a quarter inches. Good local and general condition. Patient works in a tin factory eleven hours a day, and earns five shillings and sixpence a week.

CASE 10.—Mrs. P., æt. 34. Mr. Golding-Bird, 266, 1896. Acute arthritis of hip, followed upon a miscarriage in 1896. Abscess-formation occurred. Head of femur excised on May 28th, 1896. Complete healing by June 30th,

1896. Examined May, 1904, eight years after operation. There is two inches shortening, due to slipping up of great trochanter. Great trochanter does not rise when the weight of the body is borne upon the excised joint. The joint is fixed and useful. Wasting of the thigh amounts to one inch, and of leg to one and a half inches. There is no spinal curvature. Good local and general condition. Patient performs housework; she has had one child since the operation.

CASE 11.—Charles M., æt. 11. Sir Henry Howse, 83, 1897. Three years' history of hip disease. Abscess-formation anteriorly in upper part of thigh. Excision of hip on 26th July, 1896. Sinus-formation is present. Amputation through hip-joint on 18th February, 1899. Patient made a good recovery.

CASE 12.—Frederick M., æt. 6. Mr. Lucas, 121, 1897. Sixteen months' history of hip disease. Abscess had formed on anterior aspect of upper part of thigh. On 19th November, 1896, incision of the abscess and excision of the affected hip. Healing, by primary union, complete on 27th December, 1896. Examined in May, 1904, seven years after operation. Two inches shortening, one inch being made up by slipping upwards of great trochanter, and one inch being due to shortening of leg below knee, measurements being taken from a triangle [bounded above by curved lower and internal border of internal condyle of femur; below by upper and inner edge of the internal tuberosity of tibia, and externally by inner border of patella and ligamentum patellæ], to the tip of the internal malleolus. The great trochanter does not rise when the weight of the body is borne upon the excised joint. The joint is fixed and useful. Wasting of thigh amounts to three and a half inches, and of leg to one and a half inches. There is no spinal curvature, and no sinus, but the general condition of the patient is scrofulous.

CASE 13.—George T., æt. 5. Mr. Golding-Bird, 535, 1897. Two years' history of hip disease. Excision of hip on the anterior aspect of thigh at the upper and outer part on the 31st July, 1897. Complete healing by December 6th, 1897. Examined May 23rd, 1904, seven years after the operation. Two inches shortening on affected side, due to slipping up of great trochanter. Great trochanter does not rise when the weight of the body is borne upon the excised joint. The excised joint is fixed, and useful. There is no spinal curvature. The wasting of the thigh amounts to three inches, and of the leg to one and a quarter inches. The local and general conditions are good.

CASE 14.—George N., æt. 5. Mr. Golding-Bird, 381, 1897. Sixteen months' history of hip trouble. Abscess formed over great trochanter. Excision performed on January 12th, 1897. Formation of many sinuses, which had, however, nearly healed on discharge of patient from hospital on October 25th, 1897. Examined May, 1904, seven years after the operation. Two inches shortening, made up by slipping upwards of great trochanter for one inch, and one inch shortening below knee (points for measurement as in case 12). Great trochanter does not rise when the weight of the body is borne upon the excised joint. The joint is fixed and useful. Sinuses are present over joint. Wasting of thigh amounts to two and a half inches, and

of leg to one-quarter inch. General condition is scrofulous, but not tuberculous. Bryant's double splint was used in this case, it was discarded shortly after discharge from hospital. Crutches were used for twelve months after discharge. There is very little lordosis of spine.

CASE 15.—James G., æt. 5. Mr. Golding-Bird, 213, 1897. Seven months' history of hip trouble. Abscess formed in anterior and upper part of thigh. Excision of head of femur, February 22nd, 1897. The wound healed by primary union, but patient contracted diphtheria whilst in Guy's Hospital, and was sent to a fever hospital. Mr. Golding-Bird saw the patient on June 10th, 1897, when splints were discarded. The condition noted then was, that the hip was firmly ankylosed and that there was only a quarter of an inch shortening. Examined May, 1904, seven years after operation. Patient has not used any support for two years. Two inches shortening, due to slipping up of great trochanter. Great trochanter does not rise when the weight of the body is borne upon the excised joint. The joint is fixed, and useful. The thigh is wasted, being two and a half inches less in circumference than the other one. Wasting of the leg amounts to three-quarters of an inch. No spinal curvature. General condition is poor, the child looking very delicate. There is an abscess pointing under the scar, which is stretched, but not yet broken. This abscess, according to the parents, is the first sign of recurrence of disease since the operation.

CASE 16.—Gladys C., æt. 11. Mr. Symonds, 1902. Five months' history of hip trouble. Excision of head of femur on the 20th of June, 1902. Caries of acetabulum with an intrapelvic abscess was discovered. Sinuses formed after operation, but healing had taken place by August 1st, 1902. The parents informed the writer in December, 1903, that sinuses were present and that there was about two inches shortening.

CASE 17.—John D., æt. 5. Mr. Symonds, 1902. First symptoms of hip disease are said to have appeared in March, 1902. On May 5th, 1902, an abscess formed over great trochanter, and so, shortly after this date, the head of the femur was excised: sinuses formed, and were continually being scraped. In October, 1902, it is recorded that there was one inch shortening. Examined July, 1904, one year after operation. One inch shortening due to displacement upwards of great trochanter. Joint is fixed and useful. Wasting of thigh amounts to one and a half inches; there is no wasting of the leg. There is no spinal curvature, good general condition, but sinuses are still present over excised hip. Patient still uses crutches, and a Thomas' single hip splint.

CASE 18.—Frederick O., æt. 3. Mr. Symonds, 19, 1903. Excision of head of femur on November 20th, 1902. Hoffa's plaster splint, Liston's long splint and Thomas' double splint were applied in succession. Patient discharged on February 12th, 1903, with no splint and with half an inch shortening. Examined July, 1904, twenty-one months after operation. One inch shortening, due to displacement upwards of great trochanter. Great trochanter does not rise when the weight of the body is borne upon the

excised joint. The joint is movable and useful. Wasting of thigh amounts to one inch. There is no wasting of the leg, and there is no spinal curvature. Good local and general condition; there are no sinuses.

CASE 19.—Fred C., æt. 5. Mr. Jacobson, 20, 1902. Four months' history of hip trouble. Abscess formed over great trochanter. Hip excised on the 29th September, 1899. Thomas' splint was used till July, 1903. Sinuses formed and persisted: there was a fixed joint. Amputation, through hip joint, July 20th, 1901. Patient was discharged on January 29th, 1902, having made a fair recovery.

CASE 20.—Eliza H., æt. 5. Sir Henry Howse, 42, 1899. In 1880 excision of hip performed for hip disease; abscesses and sinuses continually forming and being treated in a conservative manner. Amputation through hip was performed in January, 1899 (nineteen years after previous excision). Patient was discharged, practically well, in October, 1899.

CASE 21.—Joseph C., æt. 7. Mr. Davies-Colley, 259, 1896. Excision of hip for tuberculous hip disease with abscess but no sinus-formation in 1891. Abscesses and sinuses persisted. Lardaceous disease set in. Amputation through hip-joint performed in 1896. Patient died October 18th, 1897.

CASE 22.—Walter W., æt. 35. Sir Henry Howse, 442, 1897. Excision of head of femur in April, 1897. Abscess-formation had taken place. June 7th, 1897, patient was discharged, having made a good recovery. On November 24th, 1903, six years after operation, sinuses were forming. Patient wears an ordinary boot, with the heel raised an inch.

CASE 23.—John R., æt. 14. Mr. Lucas, 115, 1896. Two years' history of hip trouble. On October 6th, 1896, excision of head of femur was done; abscess found in thigh; wound healed by primary union and patient was discharged on November 13th, 1896; the excised joint being stiff. Examined May 21st, 1904, eight years after operation. Three inches shortening, two inches of this being due to slipping up of great trochanter, and one inch to shortening of leg below knee (the same points being used for measurement as in Case 12). Great trochanter does not rise when the weight of the body is borne upon the excised joint. The joint is partially movable and useful. Wasting of thigh amounts to five inches, and of leg two. Good local and general condition; no sinuses present. Patient works nine hours a day, and is earning thirty shillings per week.

CASE 24.—Dolly T., æt. 4. Mr. Lucas, 1897. No details as to history. Admitted on 20th May, 1897. Head was excised on the same day; death occurred same evening, immediately after the operation.

CASE 25.—Harriet B., æt. 43. Mr. Lucas, 2, 1898. Hip disease had existed for 28 years. Abscesses and sinuses had continually been forming, but for three years previous to admission there had been no abscess nor sinus-formation. On October 15th, 1897, the head of the femur was excised, and "en route" an abscess was opened. Patient's wound was not closed on January 31st, 1898; death, from husband's information, occurred in 1900.

CASE 26.—Augusta C., æt. 4. Mr. Symonds, 1890. One year's history of hip trouble. Excision on March 25th, 1890, when an abscess was found, but no sinuses; healing took place by primary union, and patient was discharged on April 26th, 1890. A Bryant's double splint was used for fourteen months after discharge, then Thomas' single splint was used for six months. Examined in June, 1904, fourteen years after excision, three inches shortening, two inches of this being due to rising of great trochanter, and one inch due to shortening of leg below knee (measurements as in Case 12). Great trochanter does not rise when weight of body borne upon excised joint. Joint fixed and useful. Wasting of thigh amounts to three inches, and of leg to two inches. There is so much tilting upwards of pelvis on the affected side, that patient wears a boot with a nine inch heel, and there is lordosis amounting to thirty degrees of flexion in hip-joint. Good local and general condition. No trace of sinus-formation.

CASE 27.—Ellen R., æt. 16. Mr. Symonds, 1903. One year's history of hip disease. Spinal caries also present. Excision of hip in 1902; intra-pelvic abscess found; sinus-formation occurred. In middle of 1903 lardaceous disease set in and amputation through hip-joint was performed; sinuses persisted. Patient discharged, and was last heard of in October, 1904, when her condition was improved.

The third group, Nos. 28 to 36, comprises those cases in which excision was performed after abscess and sinus-formation had taken place. There were three successes, in two cases (30 and 31) with fixed joints, and in one, case (32) with a movable joint; two failures, both having fatal termination, and four partial failures, three with movable joints (cases 28, 29 and 34); case 28 showed a flail joint. The other partial failure, case 33, shows no record as to mobility or fixity:—

CASE 28.—James William B., æt. 5. Sir Henry Howse, 58, 1897. Two years' history of hip disease. Abscess and sinus-formation. Excision of hip on 12th March, 1897. Complete healing on 31st December, 1897. Examined in December, 1903, six years after excision. Four inches shortening of affected limb, three inches being due to displacement upwards of great trochanter, and one inch to shortening of leg below knee (measurements as in case 12). Great trochanter does not rise when the weight of the body is borne upon the excised joint; the joint is flail and is of no apparent use to the patient. There is slight lordosis. Talipes equinus present, and sinus-formation continually recurring. Patient still uses crutches.

CASE 29. Fred J., æt. 51. Mr. Lucas, 315, 1902. Three years' history of hip trouble. Sinus-formation had occurred. Excision of head of femur in May, 1902. Wound completely healed by granulations in July, 1902. Examined May, 1904, two years after operation. Two and a half inches shortening of affected limb, due to slipping upwards of great trochanter.

Great trochanter does not rise when weight put upon it, but weight of body cannot be borne upon excised joint. The joint is movable in all directions. The general condition of the patient is good. Sinus continually forming, healing and re-forming. Wasting of thigh amounts to two inches, and of leg to three-quarters of an inch; there is no spinal curvature. Patient does no work, but obtains eight shilling a week from clubs.

CASE 30.—John B. A., æt. 13. Sir Alfred Fripp, 99, 1900. Four months' history of hip trouble, possibly pyæmic in origin. Sinus-formation took place. Excision of head of femur in August, 1901. Discharged practically well on October 12th, 1900. Examined May, 1904, four years after excision. There was four and a half inches shortening, three and a half inches being due to slipping up of great trochanter, and one inch due to shortening of leg below knee (measurements as in case 12). Great trochanter does not rise when weight of body is borne upon the excised joint. The joint is fixed and useful; no sinuses present. The wasting of thigh amounts to one and a half inches, and of leg to only a quarter of an inch. There is no spinal curvature of a permanent nature. Patient used crutches for one and a half years after his discharge from the hospital. This boy is healthy, well developed, but short. Two of his brothers were also well developed, but well over six feet in height.

CASE 31.—Harry Y., æt. 12. Mr. Symonds, 289, 1903. Uncertain history, but in 1902 sinuses formed in connection with hip disease. The head of the femur was excised in May, 1903; sinuses had completely healed by August, 1903. Patient discharged in 1903. Examined by Mr. T. C. Lucas, R.A.M.C., May, 1904. Three-quarters of an inch shortening in affected limb. The excised joint is flexed very much. There is some lordosis of the spine. The joint is fixed and useful. Wasting of thigh amounts to two and three-quarter inches. There is no wasting in the leg; general condition is good.

CASE 32.—Elizabeth B., æt. 7. Mr. Symonds, 1902. Nearly two years' history of hip disease. Abscess and sinus-formation occurred. Head of femur excised in October, 1902. Examined in July, 1904, twenty months after excision. Two and a half inches shortening, due to displacement upwards of great trochanter. Great trochanter does not rise when the weight of the body is borne upon the excised joint. The joint is movable and useful. There is no special curvature. Wasting of thigh amounts to half an inch, and of leg to a quarter of an inch. No sinuses present; good general condition.

CASE 33.—Wm. T., æt. 12. Mr. Golding-Bird, 287, 1897. Ten months' history of abscess and sinus-formation in connection with hip trouble. Excision of head of femur on October 4th. Death from shock on same night.

CASE 34.—Keith H., æt. 5. Sir Henry Howse, 27, 1902. One year's history of hip disease, with three months' history of abscess and sinus-formation. Excision of head of femur April 1st, 1902. Patient discharged on May 3rd, 1902, with wound partially healed. Examined July, 1904, two years after excision. There is one and three-quarters of an inch shortening, due to slipping upwards of great trochanter. Great trochanter does not rise when weight of body is borne upon the excised joint. The joint is movable and

useful. Wasting of thigh amounts to three and three-quarter inches ; wasting of leg, two and a half inches. No obvious spinal curvature. The boy is delicate. Sinuses continually forming, healing and re-forming.

CASE 35.—Wm. C., æt. 8. Mr. Davies Colley, 1, 1899. Admitted in September, 1898, with double hip disease and extensive abscess and sinus-formation on the left side. Excision of head of right femur on the 18th October, 1898 Sinuses persisted. Lardaceous disease set in, and patient died on April 30th, 1899.

CASE 36.—Wm. T., æt. 19. Mr. Lucas, 1902. Two years' history of hip disease. Abscess formed and sinus appeared three weeks previous to excision of hip on May 12th, 1902. May, 1904, patient wrote a letter to say he was doing work as night watchman ; his general health was good, but sinuses were continually forming, healing and re-forming.

In the following group the history of the cases is imperfect :—

CASE 37.—Minnie G., under care of a Russian Surgeon ten years ago. Excision of hip-joint ten years ago, *i.e.*, 1894. Examined July, 1904, when under care of Mr. Steward. Two inches shortening on affected side, due to slipping up of great trochanter. The whole weight of the body can be borne upon the excised joint without the trochanter being displaced upwards. There is marked flexion of the excised joint. Local and general conditions are good. Flexion has been overcome by an operation performed by Mr. Steward.

CASE 38.—Harry A., 14, out-patient, under care of Mr. Steward. Operation of excision of hip in 1901. Examined July, 1904, three years after operation. Three inches shortening on affected side, due to slipping upwards of great trochanter. Excised joint cannot support weight of body. Good local and general condition. Wasting of thigh amounts to three and a half inches ; wasting of leg amounts to two inches ; no lordosis.

CASE 39.—Margaret A. A., æt. 13. Sir Henry Howse, 164, 1898. Excision had apparently been performed at Leicester some time previous to amputation through hip-joint by Sir Henry Howse in 1898.

CASE 40.—Samuel C. T., æt., 18. Sir Henry Howse, 40, 1901. No history given by clerk in notes previous to excision on the 16th September, 1897. Abscess and sinus-formation had occurred some time before excision. Multiple sinuses formed about the hip, which was freely movable. An amputation was performed through the hip-joint in 1901, four years after excision, and in December, 1903, the boy was remarkably well.

364 *A Collection of Cases in which the Operation of Excision of the Hip-joint has been performed for Disease of the Joint.*

TABLE showing period of time between Excision of the Hip-joint and Amputation through the Hip-joint, or Death :—

	Interval.				Result.
CASE 2.	Two years	Death.
6.	Seven years	Amputation.
7.	One year	"
11.	Two and a half years	"
19.	Two years	"
20.	Nineteen years	"
21.	Five years	...	Amputation ; death in following year.		
24.	Immediately after operation	Death.
25.	Three years	"
27.	Six months	Amputation.
33.	Immediately after operation	Death.
35.	Six months	"
89.	Uncertain	Amputation.
40.	Four years	"

The writer owes a debt of gratitude to the surgeons at Guy's Hospital for their kindness in allowing him to utilise their cases, and especially to Mr. Steward for his suggestions, also to Mr. F. H. Fuller for his kindness in tracing certain cases.

NOTES FROM THE ACTINOTHERAPEUTIC DEPARTMENT AT GUY'S.

A YEAR'S EXPERIENCE.

BY G. SICHEL.

THIS Department is at present in its infancy. The first case, a recurrent carcinoma mammae, began treatment with X-rays on 18th August, 1903. Up to August 5th, 1904, sixty-one patients have attended the department. These sixty-one cases were as follows:—

Malignant disease	27
Rodent ulcers	8
Lupus	12
Mycosis fungoides	1
Glossitis	2
Chronic rhinitis	2
Lupus erythematosus	1

53

This leaves eight cases unclassified which will be shortly referred to at the end of this paper.

MALIGNANT DISEASE.

CASE 1.—N. S., female, æt. 43. Recurrent carcinoma of left breast. She was operated on two years ago by Sir A. D. Fripp. A small lump appeared again seventeen months after; four months ago began X-ray treatment elsewhere, which had to be stopped for six weeks on account of X-ray burn. Treatment here extended from 18th August, 1903, to 30th October, 1903. Twenty-five sittings of X-rays for at first ten, later fifteen minutes. Cossor's tube was used. Twenty-six sittings of the static breeze, fifteen minutes each, from a large Wimshurst machine.

Remarks.—The X-rays and static breeze were applied alternately in the hope of preventing the recurrence of an X-ray burn. Result.—No increase of tumour in two months; no pain. No cure, but alleviation. Patient left to return to her home in Norfolk and promised to attend Norwich Hospital for further treatment. I wrote to the house-surgeon of Norwich Hospital in July last, but he told me she had not been attending there.

CASE 2.—C. P., male, æt. 69. Extensive recurrent epithelioma of mucous membrane of mouth and right cheek, no enlarged glands. Treatment lasted from 20th August, 1903, to 23rd December, 1903, when he had to give up coming from weakness. X-rays, forty-two sittings; static breeze thirty-three times. The static breeze was applied when redness of the skin (? from the X-rays) threatened dermatitis. A large Cossor's tube was used, and on sixteen occasions, with the X-rays externally, 5 mgms. of radium bromide in a glass tube were applied inside the mouth. As far as I could judge, he did not benefit in the slightest and the treatment was a complete failure.

CASE 3.—G. D., male, æt. 54. Carcinoma of the lower jaw. He had been operated upon six months previously. Came up in an almost hopeless condition. Only attended four sittings of X-rays with Cossor's tube and then ceased to appear. Three of the sittings lasted twenty minutes, and one fifteen minutes. There was reddening of the skin and apparently some softening of the anterior part of the growth. There was no benefit to the patient, but the tumour was far too advanced and the treatment far too brief to expect any.

CASE 4.—E. M., an old Welsh woman who could not speak English. Epithelioma of right side of scalp which began five years ago. Very foul; a few cervical glands enlarged. Treatment began on 31st August, 1903, and continued until 16th November, 1903. Thirty-seven sittings of X-rays with large Cossor's tube, generally for fifteen minutes. Treated seventeen times with the negative static breeze.

Remarks.—W. B. Hardy and Miss E. G. Willcock found that in certain experiments the activity of radium emanations was increased in the presence of NaCl, so I used to sponge over the growth with a strong solution of common salt before treatment. The disease was naturally slow in growing, and I do not believe she received the slightest benefit from the treatment. She left at her own wish to go home. The epithelioma had spread and the glands in the neck had broken down.



Taken 8th October, 1903. **FIG. 1.**



Taken 15th February, 1904. **FIG. 2.**

CASE 5.—J. K., male, æt. 46. Lymphosarcoma of the glands of the neck, which had been operated upon twice. The growth presented a large oozing fungating mass protruding behind the angle of the jaw. Treatment:—Eleven X-ray sittings with Cossor's tube, fifteen minutes each, five sittings with static breeze. As there was no benefit, and the sarcoma increased, he gave it up to submit to a third operation.

CASE 6.—A. W., female, æt. 70. Recurrent carcinoma of the breast. Mr. Lucas had removed the left breast four years ago. The recurrent growth was extremely painful and tender, and the left arm was swollen. Treatment lasted from the 16th September to the 31st December, 1903. X-rays (Cossor's tube), sixteen times, of ten to fifteen minutes each sitting. Static electricity, negative breeze, for fifteen minutes, forty-three times. Positive charges, ten times.

Remarks.—Pain was much alleviated in this case, and she was certain she got more relief from the static electricity than from the X-rays. Nevertheless she steadily became worse and had to desist from attending owing to left pleuritic effusion, due to extension of the growth.

CASE 7.—S. M., female, æt. 50. Inoperable carcinoma of cervix uteri sent by Dr. Horrocks. Treatment.—Six sittings of X-rays by small Cossor's tube.

Remarks.—No benefit, and patient too ill to continue treatment.

CASE 8.—L. H., female, æt. 63. Sent by Mr. Dunn. Began twenty-six or twenty-seven years ago as a small wart. Has been operated upon. Came here with what looked like a rodent ulcer of the right cheek, lip and ala of nose. Treatment lasted from 8th October, 1903, to 12th February, 1904. Fourteen sittings of X-rays with Cossor's tubes. Eleven sittings of static negative breeze. Forty-six sittings of 5 mgms. of radium bromide. Remarks.—Entire failure to do good; ulcer increased in size (see photographs) and continued extremely tender and painful throughout. At times it appeared to be commencing to heal and then all would break down again. The radium bromide was supplemented by the NaCl treatment mentioned in case 4, also by applying oxygen from a cylinder over the ulcer, on the supposition that the action of radium depended upon the presence of this gas, and would therefore be more efficient in the presence of an excess. I also painted over the surface of the ulcer with a solution of methyl blue in collodion, on the supposition that the rays at the red end of the spectrum might be harmful. However, I afterwards found with the spectroscope that my solution painted on glass did not prevent the red rays coming through. Towards the end the case had more the appearance of an epithelioma than a rodent ulcer, and as such it is classified here. (Figs. 1 and 2.)

CASE 9.—J. K., æt. 65, sent here for treatment for carcinoma of the cervix uteri after operation by Mr. G. B. Smith. Treatment lasted from 25th September, 1903, to 6th October, 1903. Seven sittings with X-rays (Cossor's tube), in all one and a half hours. On 6th October examined by Dr. Horrocks and Mr. G. B. Smith and no growth could be found. There was no further treatment, as patient was very sore after examination, and on 12th October she was discharged to a convalescent home.

Remarks.—I am indebted to Mr. Hicks, the Obstetrical Registrar, for further information respecting this case. She was re-admitted into Patience ward under Mr. G. B. Smith from November 28th to December 13th, 1903. There was undoubted growth of the cervix, and a piece removed showed that it was an epithelioma. She never came to my department again.

CASE 10.—L. M., female, æt. 29. Carcinoma of the cervix, sent for treatment after operation on 14th October, 1903. Treatment commenced 26th October, 1903, and went on till 8th February, 1904. Thirty sittings, fifteen minutes, X-rays, with small Cossor tube.

Remarks.—Absolute failure to do any good with either pain or growth.

CASE 11.—E. M., female, æt. 25. Inoperable case of carcinoma of the cervix uteri. Treatment lasted from 29th October, 1903, to 11th January, 1904. Thirty sittings of X-rays (small Cossor's tube), fifteen minutes each.

Remarks.—Absolute failure to do any good.

CASE 12.—H. W., female, æt. 45. Inoperable carcinoma of the cervix uteri, sent by Dr. Horrocks. Treatment from 23rd November to 23rd December, 1903. Ten sittings of X-rays (Cossor's tube).

Remarks.—Absolute failure to do any good; patient had to give up attending owing to excessive weakness.

CASE 13.—M. B., female, æt. 69. Epithelioma of the cervix uteri operated upon by Dr. Horrocks thirteen days before she was sent here. Treatment from 21st December, 1903, to 5th January, 1904. Nine sittings of X-rays, (Cossor's tube), of fifteen minutes each. No benefit from treatment.

CASE 14.—M. C., female, æt. 44. Sent by Dr. Horrocks. Carcinoma of the cervix uteri. Operation in August and again in December, 1903. Recurrence. Treatment commenced on 23rd December, 1903, and lasted till 26th April, 1904. X-rays, forty-three sittings of fifteen minutes. Acting on Dr. Horrocks' suggestion this treatment was supplemented on eighteen occasions by the action of radium.

Remarks.—Probably some alleviation. When the radium was applied as well, the X-rays were applied externally, with 5 mgms. of radium bromide in a glass tube in the vagina, and on sixteen occasions there was a radioactive screen on the back, in the lumbo-sacral region as well. I am afraid that this case also must be looked upon as a failure.

CASE 15.—G. H., male, æt. 53. Recurrent epithelioma of the right cheek, very extensive and inoperable. Treated from 12th February to 25th April, 1904. X-rays from a large Cossor's tube, thirty-five sittings of fifteen minutes. No benefit whatever.

CASE 16.—E. H., female, æt. 62. Inoperable carcinoma of the cervix uteri. Treated with X-rays, small Cossor tube, in vagina from 15th February, to 19th May, 1904. Forty-two sittings of fifteen minutes.

Remarks.—Absolutely no beneficial effect on growth. She told me she was never free from pain except during the actual application of the rays. I last saw her on 13th June; she was very ill, much emaciated, and in great pain.

CASE 17.—E. K., female, æt. 45. Recurrent epithelioma of the vulva. Nine sittings of radium bromide for fifteen minutes, except the first, which was only five minutes. Patient did not persevere with the treatment and left in statu quo. She attended from 29th February to 7th March, when she ceased attending owing to menstruation, and again from 21st to 31st March.

CASE 18.—M. W., female, æt. 61. Inoperable carcinoma of the cervix uteri. Attended from 10th March to 7th April. Fifteen sittings of X-rays for fifteen minutes each. Small Cossor tube used per vaginam. No apparent benefit.

CASE 19.—M. S., female, æt. 60. Recurrent carcinoma of the breast sent to me by Mr. Rowlands. Treatment lasted from 14th March to 7th April, during which time she had thirteen sittings of X-rays lasting fifteen minutes each.

Remarks.—At the end of the treatment Mr. Rowlands took her into Bright ward to remove a mass of glands in the neck. Both he and the patient were of opinion that the recurrence which had been treated with X-rays had got smaller. The tube used was a hard one, averaging a six-inch alternative spark. Deane-Harnack's apparatus was used.

CASE 20.—A. H., female, æt. 37. Scirrhus of the left breast in such an advanced stage that she could hardly stand alone. Given X-rays for fifteen minutes on one occasion; she never attended again.

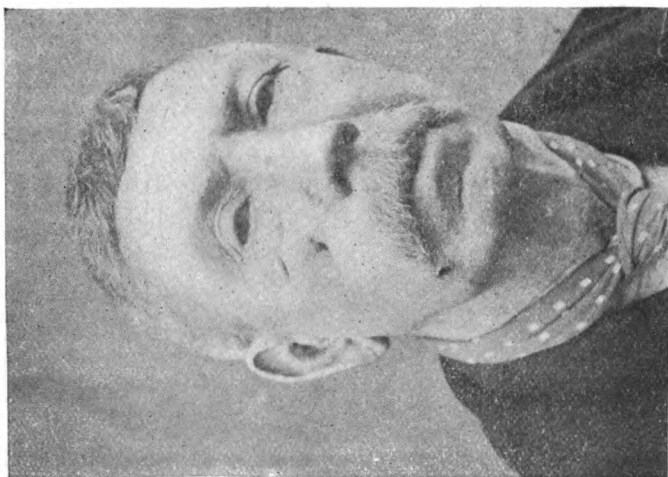
Remarks.—This is an example of the hopeless condition in which many cases are when they are sent for X-ray treatment as a dernier resort. They are not fair tests from which any reliable deductions can be drawn.

CASE 21.—F. B., female, æt. 38. Inoperable carcinoma of the cervix uteri. Seven sittings with X-rays for ten minutes each, except the last, which was fifteen minutes. She said she felt better, but ceased to attend, so do not suppose she felt any real benefit.

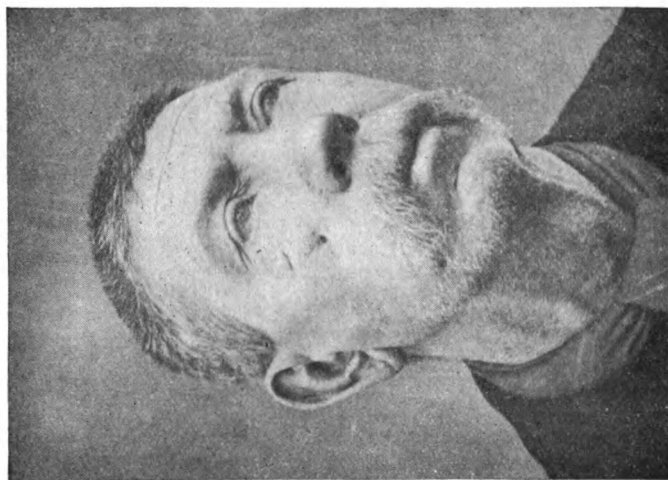
CASE 22.—L. T., male, æt. 54. Recurrent epithelioma of the sole of the left foot. 13th February 1904, admitted under Mr. Lane with a fungating ulcer, two and a half inches by one and a half inches, which began in an old scar two years before. The skin behind the ulcer was covered with scabs. Numerous hard but painless glands in left groin. On 17th February the mass was removed and skin grafting was performed. 8th March, slight recurrence of growth in two places. 22nd March to 5th May, X-ray treatment, twenty sittings of fifteen minutes. Ulcer healed up, but the raised indurated border remained. On 9th April two bits were removed and found by the pathologist to be still epitheliomatous. He went home, to Wales, and so treatment was discontinued.

Remarks.—I think that in this case the rays had decidedly a beneficial influence, but in view of the microscopical examination it is more than probable that the improvement observed will be only temporary.

CASE 23.—C. S., female, æt. 54. Recurrent carcinoma mammae. Had been operated upon twice for carcinoma of the right breast. Only attended four times for X-rays. It is useless to try and draw any deductions from this case.



Taken 14th October, 1903.
FIG. 3.



Taken 4th January, 1904.
FIG. 4.

CASE 24.—M. E., female, æt. 49. Sent by Mr. Jacobson. Right breast was removed for carcinoma about eighteen months before; now has a recurrence about the size of small orange in the scar and attached to the ribs. Treatment commenced on 28th April, and is still continuing at the time of writing, 7th August, 1904. Forty-three sittings with X-rays usually of ten minutes duration. On 24th June, Mr. Jacobson was kind enough to take her into Dorcas ward to keep her under observation; when the effect of embedding 5 mgms. of radium bromide in a glass tube, into the centre of the growth was tried, it was put in in three different places by Mr. Adams (Mr. Jacobson's dresser) and finally removed on 15th July, when the X-ray treatment was continued. With the exception of easing the pain, of which the patient is certain, there is apparently no benefit. The growth is much larger than it was and patient, who is most cheerful and hopeful, is becoming cachectic.

CASE 25.—S. W., female, æt. 47. Extensive carcinoma of the cervix uteri which had been operated upon and partially removed. Attended four times for X-ray treatment and then did not appear.

CASE 26.—D. W., female, æt. 67. Atrophic scirrhus, left breast, sent to me by Mr. Steward. She has only noticed it a year, but thinks she has had it longer. The nipple is much retracted, and there is a puckered-in, red, linear, ulcerating scar, deeply attached, in the lower part of the left breast. Treatment commenced on 20th June, 1904, and she is still attending (7th August). She has had twenty-two sittings of the X-rays lasting ten minutes each. On the whole, there is some improvement locally; there is less discharge, and the growth is at all events no larger than it was; on the other hand, I am afraid the patient is slowly losing flesh.

CASE 27.—E. S., female, æt. 50. Recurrent carcinoma mammae. Has been operated upon three times in three years. Multiple carcinomatous nodules of the skin, sent by Mr. Lucas on 14th July, 1904; still under treatment, 7th August, 1904. Has had eleven sittings of X-rays usually ten minutes each. The largest nodule, which is ulcerating, has been picked out for treatment. No change so far.

Conclusions as regards malignant disease.—Of the above twenty-seven cases, eight (viz., 3, 5, 7, 9, 12, 20, 23, and 25) may be disregarded as being of any value, since either from being in too advanced a condition, or from want of immediate alleviation, their attendances were too meagre to prove the value or otherwise of the treatment. And here we must remember that it is practically only inoperable cases which have been sent to this department. Of the other nineteen cases, there was some alleviation (i.e., relief of pain) and perhaps some retardation of growth in five (viz., 1, 6, 14, 19 and 22). About two (26 and 27) I would give no opinion at present, and they are still under



Taken 29th January, 1904. FIG. 6.



Taken 10th December, 1903. FIG. 5.

treatment; the other 12 must, I regret to say, be recorded as absolute failures.

As regards the details of treatment, I have always used high tubes, which accounts for the fact that no case of dermatitis has occurred. Several cases of decided reaction (*i.e.*, redness and loss of hair) have occurred, but the appearance has soon subsided on the cessation of treatment. The current used has varied from four to six ampères at about ninety volts. The alternative spark gap of the tubes which have been used is about six inches.

The results cannot be looked upon as very hopeful, perhaps lower tubes, which I now intend to try, will give better results, at the same time, as the cases sent were practically hopeless, it is well to remember that in about one case out of every three one may hope to lessen pain or retard growth for a time. It is interesting to note that in case 6 pain was more relieved by static electricity than by the X-rays.

RODENT ULCERS.

CASE 1.—J. L., male, æt. 72. Rodent ulcer of the left temple, had been operated upon six times. Treatment: five sittings of X-rays, one of the static breeze, after which treatment was discontinued and he was operated upon by Mr. Jacobson.

CASE 2.—G. T., male, æt. 65. Rodent ulcer over the right lower jaw; another situated higher on cheek had been operated upon (see Figs. 4 and 5). Forty-two sittings of 5 mgms. radium bromide in a glass tube. Treatment lasted from 14th October to 31st December, 1903. A depressed scar was left. He was last seen on 28th July, 1904. There was no recurrence. The treatment was generally supplemented by a preliminary bathing with NaCl solution and the ulcer painted over with a solution of methyl blue in collodion, but I do not think these preliminaries can have been of much help (see Figs. 3 and 4).

CASE 3.—L. E., female, æt. 38. Small rodent ulcer near the inner canthus of the right eye (see Figs. 5 and 6). Had twenty applications of radium bromide 15 mgms. in glass tube, fifteen minutes at a time. Treatment lasted from 9th December, 1903, to 25th January, 1904. Ulcer healed leaving a soft, slightly pink scar. I again saw her on 11th March, when there was some very slight thickening to be seen at the upper part of the scar. A slight warty lump subsequently formed here, which was again treated on 22nd and 29th April, and 6th and 13th May, 1904, when it had become much smoother and she discontinued her attendance. Thirty-minute sittings were given on the last three occasions.



Taken 7th October, 1903. FIG. 7.



Taken 9th March 1904. FIG. 8.

CASE 4.—G. C., male, æt. 71. Rodent ulcer the size of a shilling just in front of the right ear. Attended for seven applications and then discontinued coming. The ulcer was beginning to improve. He was given X-rays for fifteen minutes a time.

CASE 5.—M. G., female, æt. 58. Small rodent ulcer of the left upper lip. Treatment from 3rd May to 15th July with a compound salt of radium and barium of seven thousand radio-active units, one dec'grm, behind a mica shield in a vulcanite holder, thirty-five sittings of thirty minutes each. After nine sittings the ulcer appeared almost healed and then in spite of treatment relapsed and even got a little bigger. From 18th July to the present time radium bromide 5 mgms. in a glass tube has been used and the ulcer appears to be slowly healing; some redness of the surrounding skin has been caused. Patient is not anxious to have an operation. She has had in all thirty-five applications of radium and barium and eleven of radium bromide. So far the case must be looked upon as a failure, and if the patient consents I shall discontinue treatment and recommend her for operation.

CASE 6.—A. G., female, æt. 69. Multiple rodent ulcers of the right cheek. Operated upon four years ago. Has had four applications of radium bromide and is still under treatment.

CASE 7.—T. M., male, æt. 60. Rodent ulcer of the right cheek. Was operated upon five years ago. Has had eight applications of X-rays of ten minutes each; is still under treatment. Patient says there is less discharge.

CASE 8.—W. J., a middle aged Welshman, who can only speak a little English. Rodent ulcer attacking nose. Began treatment on 16th June, and is still under treatment. Twenty-two sittings with X-rays. The ulcer appears to be spreading in one direction and healing in another. Treatment changed to radium bromide, of which he has only had one sitting at the time of writing (7th August). If in another week the spreading on the one side still continues I shall discontinue the treatment and send him to one of the surgical staff with a view to operation.

Conclusions as regards rodent ulcer.—Out of the eight cases three are still under treatment (6, 7 and 8) and two (cases 1 and 4) failed to persevere with it. This leaves three cases for consideration; of these I think two (cases 2 and 3) may be considered successful, and one (case 5) a failure. These were all treated with radium salts, and I think the failure may be perhaps due to inefficient therapeutic value in the salt used, or to the fact that besides the mica shield I also used a thin sheet of gutta-percha tissue to prevent the radium getting spoilt by moisture from the skin. Rodent ulcer has been shown to be amenable to X-rays

in other people's hands, but I have not been able to give them a fair trial yet, except in case 8, where they failed. Radium is useful, because it could be applied at a patient's own home.

LUPUS.

CASE 1.—L. S., female, æt. 21. A small, recurrent, but very thick and obstinate patch on the upper lip (see Figs. 7 and 8). Treatment lasted from 18th September, 1903, to 7th March, 1904. She had in all one hundred and five sittings, of which forty-four were X-rays, using Cossor's lead-glass tubes; six radium bromide, and fifty-five ultraviolet light from an iron spark lamp; in the last fourteen ultraviolet sittings L. Miller's lamp was used, but the previous application had been by a very inefficient instrument by another maker. The sittings were usually fifteen minutes each and only once was any reaction obtained. At the end the hard lumps were touched with pure carbolic; this was done twice. A pale soft scar was left. At various times she was prescribed Iron, Arsenic, Potassium Iodide, Perchloride of Mercury, and Extract of Malt. Locally Ung. Picis Carbonis. I had a letter nearly two months after she left to say that the cure persisted.

Remarks.—The treatment was unduly long, a great deal I think on account of the inefficient instrument used before the Leslie Miller lamp.

CASE 2.—M. M., female, æt. 31. Extensive lupus on the chin, right cheek and right ear with superficial ulceration and much scabbing. Had been treated with X-rays from July to November, 1902, and discharged as almost cured; it recurred and was again treated with X-rays from March, 1903, to February, 1904, with absolutely no benefit. She was then sent here for the light treatment. Treatment began on 8th February, 1904, and has continued to the present time. She has had in all fifty-four sittings, all with the Leslie Miller iron spark lamp, except one with radium and one with the Lortet-Genoud modification of Finsen's lamp. She is nearly cured at the time of writing, 7th August, 1904, and what was an unsightly ulcerating area, is now smooth scar tissue with a few scattered apple-jelly nodules here and there.

CASE 3.—J. F., male, æt. 22. Lupus all over the front of his chest, the right side of the face and left cheek. Sent up by Dr. J. Richards. I failed to do the slightest good in this case, either with light or X-rays. He attended from 5th January to 10th May, 1904, and had in all eighty-four sittings. Once radium was applied. I tried Leslie Miller's lamp, another iron spark lamp, a carbon arc lamp, kindly lent me for trial by the Dowsing Company, and Cossor's tubes for the X-rays. We had not the proper Finsen lamps installed at this time. As before stated, the treatment proved an utter failure, although some reaction was obtained on several occasions.

CASE 4.—H. P., female, æt. 17. Lupus non-exedens of right thigh, lupus exedens of right foot, of which there was much deformity with loss of the big toe. Treatment commenced on 28th March, 1904, and has continued to the present time. Sixty-two applications to thigh, all, except three, ultraviolet lamp, Miller. Sixty-one applications of X-rays to foot. Besides this, on

two occasions very hard nodules in the thigh were touched with pure phenol. There is great improvement in the foot and a good deal in the thigh. The sittings for both foot and thigh were usually fifteen minutes. The case is still under treatment. Much benefit has accrued, and I hope to effect an entire cure.

CASE 5.—M. F., female, æt. 21. Lupus non-exedens of right cheek. Had been operated upon. Treatment commenced on 25th April, 1904, and is still being continued. She has had thirty-five sittings with Miller's iron spark lamp, varying from three to ten minutes, and twelve with the Lortet-Genoud lamp of thirty minutes each. There is great improvement, and a soft pliable pale scar is replacing the lupoid tissue.

CASE 6.—C. R., male, æt. 20. Lupus of the skin in front of the neck, patch rather larger than a five-shilling piece. Treatment began on 18th May, 1904, and still continues (7th August, 1904). Thirty-five sittings with Miller's lamp. Improvement is taking place, but slowly, probably because the soft tissues of the neck prevent sufficient pressure being used to produce proper anæmia of the part.

CASE 7.—A. C., female, æt. 50. Extensive lupus of the face; a good deal of destruction of the nose; ulceration. Treatment began on 8th June, and is being continued. Seventeen sittings of ten or twelve minutes with X-rays, four with the iron spark lamp, six to ten minutes, twelve with the Lortet-Genoud lamp of thirty minutes each. There is marked improvement. The X-rays were used in the ulcerated parts which were too tender to stand pressure.

CASE 8.—R. M., female, æt. 24. Lupus of the gums. Sent up by Dr. Mason, of Sudbury. Has been operated on for lupus of face, nose, and lip, seven times. This was finally cured at the London Hospital after fifteen treatments of one hour each. The present trouble of the gums has come on later. A good deal of ulceration and loss of central incisors in upper jaw. Treatment began on 28th June, and still continued. Fourteen treatments of ten minutes each with X-rays which seemed to do no good, then nine sittings with radium bromide of thirty minutes each. There is now, I think, some improvement, 7th August, 1904.

CASE 9.—F. H., female, æt. 26. Small patch of lupus on the right cheek, also a spot in the right nostril. Treatment began on 12th July and is being continued. X-rays thirteen times, ten minutes each. Miller's lamp and Lortet-Genoud lamp once each, but discontinued as efficient pressure was impossible. Great improvement.

CASE 10.—E. C., female, æt. 8. Several patches of lupus on the face and limbs. Those on the limbs are being scraped, those on the face treated with the Lortet-Genoud lamp. She has had ten sittings of thirty minutes each, and one of ten minutes with the iron spark lamp and is improving, she is still under treatment.

CASES 11 and 12.—Are of such recent date that no purpose can be served by reporting their treatment.

Conclusions as regards lupus.—Omitting the last two cases (11 and 12) I have to record entire failure to do any good in one case (No. 3), a (at all events temporary) cure in one case, marked benefit in four cases, and slight improvement in the others, some of which have not had time to show any decided change. Up to the present we have had to work with very scanty apparatus, but when the new department is fully opened our results should be much better.

MYCOSIS FUNGOIDES.

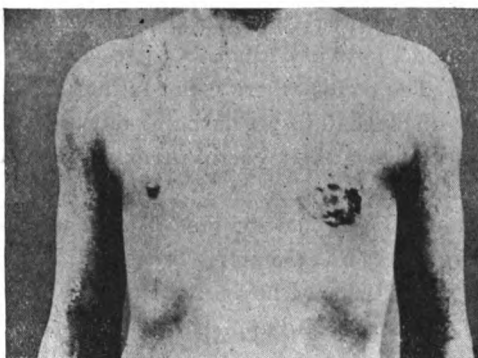
On 1st March, 1904, F. E., a strongly-built man, æt. 36, was brought to Sir Cooper Perry's out-patients, suffering from patchy, pigmented, rough blotches all over the trunk, arms and legs, with somewhat extensive ulceration about the left nipple, which was itself enlarged as a red, moist granulomatous mass, to the size and appearance of a raspberry.

Patient says the disease began in childhood. When about eight years old his mother took him to a doctor because his legs used to come out in bright red spots, especially after washing. These spots used to come and go, but gradually the skin grew rough, first on the legs and then on the arms. His father died from "influenza" at seventy-four, and his mother from "paralysis," also aged seventy-four. He has four brothers and two sisters alive and well, another brother died from "kidney disease," and another died after "operation" for (?) intestinal obstruction. He is a married man, and has two children (girls), quite healthy, and his wife is also healthy. He had "pleurisy," he thinks, on the right side, when fourteen; was troubled with varicose veins, still present, when 18, relieved by applying a Martin's bandage; "slight inflammation of the lungs" when 20. About six months ago he found a slight abrasion of the skin below and a little to the inner side of his left nipple; he treated it himself with boracic ointment for six weeks, and during this time it scabbed over, but then the scab came off, leaving an ulcer; this has gradually spread since, until it is now about the size of the palm of the hand. The skin, generally, is dry and rough, and in many places a brawny red, these brawny patches

being sometimes a slightly deeper colour at the edges, which are slightly raised. In other patches there is slight branny desquamation, and on the back of both upper arms the condition resembles ichthyosis. The face and head are free. He suffers from itching, which is evidently only slight, as there are no scratch marks; this itching has been more marked during the past few months, and is worse when he is hot or is in bed. He says his hands always swell in winter.

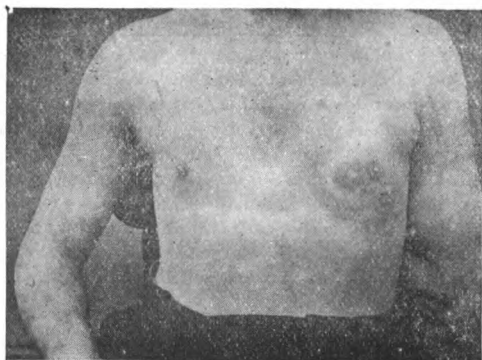
Sir Cooper Perry diagnosed mycosis fungoides, and it was resolved to treat him with X-rays. Treatment commenced on March 2nd. The X-rays were applied direct to the ulcer and fungating nipple, by means of a Deane-Harnack apparatus from a very high tube, which probably averaged about a 6-inch alternative spark gap.

The sittings on each occasion lasted fifteen minutes, except twice, when they were only ten minutes each. On the 12th April, after twenty applications, the chest was healed; patient was then found to have a small scabbed place on his back about the size of a threepenny-bit. Between 12th and 29th April this was given seven applications, and appeared healed. On 10th May, however, he came back again, the scab on the back having re-formed. He attended very irregularly until the 21st June (10 attendances), and the place on his back grew decidedly bigger. I impressed on him the necessity for regular treatment, and it has now practically healed again after twenty-three further sittings (7th August, 1904). Besides the local effects, a general improvement was clearly observed in the condition of the skin. The nipple now is normal in size and appearance, and where the ulcer was situated is now a deep pink patch of soft pliable skin, to which the term scar is hardly, perhaps, applicable. He was not given any internal medicine, and the only local application was a simple ointment and lint to protect the ulcer from pressure of his clothes. In the *Epitome of Current Medical Literature* "British Medical Journal," 9th April, 1904, is mentioned a case treated by A. E. Carrier (*Journ. Cutan. Diseases*, February, 1904), where, as here, although no cure is claimed, a distinct and remarkable effect has been obtained by X-rays in a disease which



Taken 10th March, 1904.

FIG. 9.



Taken 12th April, 1904.

FIG. 10.

is almost uniformly fatal, and for which no other treatment is known.

Scholtz, Norman Walker and H. G. Brooke have also reported good results in this affection (Freund). Radcliffe-Crocker describes three types of mycosis fungoides: I. The most common where there is some form of dermatitis antecedent to the tumour-formation. II. The rarest where recurrent attacks of lymphangitis lead up to an elephantiasic thickening of the skin, and finally tumour-development. III. Where tumours develop without antecedent dermatitis.

It is interesting to note that the present case is an example of type I., whereas Carrier's case seems to belong to type III.

I would add that the patient's private doctor, who had taken great interest in the case, had applied all the usual remedies without any good result.

I have to thank Sir Cooper Perry for his kindness in allowing me to publish these notes on his case (see Figs. 9 and 10).

GLOSSITIS.

CASE 1.—J. R., male, æt. 54. Suffering from "Sore tongue" for over a year. He has been attending a doctor for eighteen weeks. He now has a greatly thickened tongue covered with fissures and bald patches. He was sent to me by Mr. Lane, who considered the condition of the tongue was likely to end in epithelioma. The tongue was extremely painful and tender. Treatment began on 3rd March and ended on 16th May, 1904. Three sittings of X-rays of fifteen minutes each and eighteen of radium bromide. There was so much improvement that Mr. Lane advised him to discontinue the treatment. I saw him again on 13th June and 11th July, when he was still keeping well. He used a chinosol mouth wash during the treatment.

Remarks.—In view of the chronicity and possible termination of the disease in malignancy, I think this case is well worth noting.

(NOTE.—The day after writing this the patient returned complaining of his tongue being sore again. It was, however, still looking well. Treatment was recommenced.)

CASE 2.—F. N., male, æt. 31. Sent for treatment by Mr. Steward. Tongue swollen and fissured, smooth in places, superficial ulceration in others. Curious white elevated papules in parts of the ulcers, which, as Mr. Steward suggested, looked like a fungous growth, but which was examined by Dr. Eyre with negative result. The same condition existed on the palate. There is one slightly enlarged, soft, gland in the left submaxillary region. He says he has had the disease all his life. No history of syphilis; has been married eight years. Suffered from dyspepsia and measles during his childhood. Finger nails are very thin, and those of right thumb and left index

thickened as in onychomycosis. Treatment lasted from 21st June to 22nd July, 1904. Fourteen sittings of ten minutes each with X-rays. On two occasions the treatment was discontinued as the tongue got rather more sore than usual. He had to discontinue treatment in order to return to his work. The tongue was slightly improved, being not so swollen and ulcerated. This may or may not have been due to the treatment, and, as he said, his tongue varied from time to time.

CHRONIC RHINITIS.

CASE 1.—H. A., male, middle aged. Chronic, purulent, scabby, thickened condition of the upper lip, apparently dependent upon a chronic profuse, watery discharge from the nose from which he has suffered for twenty-four years. He is inclined to indulge somewhat freely in alcohol. He has been under treatment since April, 1904, and has been treated with X-rays, positive static charges, and high frequency currents; is decidedly better but is liable to relapses.

CASE 2.—L. B., female, æt. 29. Sent for treatment by Mr. Steward, on account of a chronic watery nasal discharge from both nostrils accompanied by sneezing. Has suffered from it for a year. Has been treated with high frequency currents since 31st May, 1904, and expresses herself as being very much better. She is still attending.

LUPUS ERYTHEMATOSUS.

CASE 1.—E. B., female, æt. 49. Lupus erythematosus discoides over right eye, patch about the size of a half-crown. Treated from 4th December, 1903, to 7th April, 1904, with X-rays, negative static breeze, high frequency currents and radium. It seemed at one time to improve with the static breeze—the scaliness disappeared and there was less thickening of the edges of the patch—eventually, however, after trying all the above remedies, the case had to be given up as I entirely failed to do any good, in fact she got worse instead of better.

UNCLASSIFIED CASES.

These were eight in number and will be just shortly referred to—

CASE 1.—R. D., male, æt. 25. Sent by Sir Alfred Fripp for a hard bony swelling in left occipital region, the result of an accident some time previously; inveterate headache and insomnia. Given ten sittings of the negative static breeze for fifteen minutes each. Cured of headache and sleeplessness.

CASE 2.—M. H., female, æt. 17. Small brown mole on left cheek. Given two sittings of radium and barium and two of X-rays, no change, so then advised to have it excised, which was done.

CASE 3.—F. W., male, æt. 32. For an ulcer on back of his left hand of fourteen months' duration, which followed an injury with a pair of scissors. It would not heal. Has been excised twice but recurred. Is now surrounded by a suspicious induration. Has had a course of potassium iodide. Sent to me by Mr. Dunn. Was extremely suggestive of commencing malignant disease. No microscopical examination had been made. Treatment from 16th May to 20th July, 1904, forty-two sittings with X-rays, fifteen minutes each; discharged cured. Quite healed and no induration.

CASE 4.—A. G., female, æt. 18. Old infantile paralysis of left lower extremity, trophic congestion and ulceration in small patches on toes and ankle. Treated with X-rays, galvanism and faradism. Some improvement.

CASE 5.—A. O., female, æt. 2½ years. Suffering from chronic membranous ophthalmia of the left upper lid. Had five sittings of radium bromide for five minutes each, but then developed measles and treatment had to be discontinued.

CASE 6.— — female, æt. —. Warts on hand. Attended twice for radium treatment and then discontinued coming.

CASE 7.—H. P., female, æt. 19. Sent to me as a case of lupus, as which at first I regarded it. I started treatment with a Leslie Miller lamp, and later with radium. Latterly I came to the conclusion that my diagnosis was wrong, and that it was a case of erythema dependent upon indigestion. Treated with Salol and Sodium Sulphate, and subsequently Mist. Acidi Co., with only applications of lanoline locally. She appeared to get quite well.

CASE 8.—L. L., female, æt. 30. Sent up as a case of lupus. Extensive ulceration on the back of her right leg which she has had for twenty-seven years. Has had X-rays tried for five months without any benefit. Was treated from 9th June to 18th July with first Pot. Iod. and Hyd. Perchlor., and later nerve and general tonics. Locally mercurial applications. The ulceration all healed. From 18th to 26th July, at patient's earnest request, she was given the ultra-violet light treatment from a Leslie Miller lamp. When last seen all the ulcers were healed, but a red-coppery area was left covered with scaly epithelial debris resembling ichthyosis.

NOTE.—I would like here to acknowledge my indebtedness to all the members of the Staff whose names are mentioned in the foregoing, and to Mr. Pullen, the electrician of the department, for his help, especially with the photographs, for which he is chiefly responsible.

SPECIMENS RECENTLY ADDED TO THE PATHOLOGICAL MUSEUM.

BY LAURISTON E. SHAW, M.D.,
SIR COOPER PERRY, M.D.,
AND
JOHN FAWCETT, M.D.

HEART.

2309 Imperfect Septum Ventriculorum.

The base of a heart mounted to show at the upper part of the septum a small fistulous communication between the two ventricles. The opening in the left ventricle is situated just below the attached border of the right anterior aortic cusp.

William P., æt. 19, was admitted under Dr. Pye-Smith with a cerebellar tumour, from which he died ten weeks after admission. While in the hospital a loud systolic bruit was heard over the heart, its point of greatest intensity being in the pulmonary area. At the autopsy the heart was found to weigh twelve and a half ounces, the other viscera with the exception of the brain being healthy. *See Insp.*, 1897, No. 49.

2316 Pulmonary Stenosis.

A heart divided by a vertical incision to show the aorta and pulmonary artery, both arising from the right ventricle, the wall of which is somewhat thicker than that of the left ventricle. The pulmonary valves are fused together, forming a thick membranous cone, with a central aperture not more than a line in diameter. The trunk of the pulmonary artery is small and its walls are thin. The two

ventricles communicate with each other by an opening, admitting the index-finger, at the highest part of the septum.

John V., æt. 17, was admitted under Dr. Pye-Smith for dyspnoea and attacks of syncope. On admission his lips were cyanosed, his fingers clubbed, and a systolic bruit was heard in the third left intercostal space close to the sternum. While in the hospital the patient had two epileptiform seizures, and blood and albumen were found in the urine. He died two months after admission, and at the autopsy the heart was found to weigh fifteen ounces and the kidneys were congested. *See Insp.*, 1898, No. 404.

2322 Congenital Malformation of Heart and Great Vessels.

The thoracic viscera of a fœtus mounted to show the aorta arising from the right ventricle, and giving off from its arch two vessels taking the place of the right and left branches of the pulmonary artery, the main trunk of which is absent. The descending aorta passes over the right bronchus.

From a fœtus prematurely born in Mary ward in 1888.

2323 Congenital Malformation of the Heart and Vessels.

An infant's heart mounted to show a large cavity representing the right ventricle, from which the aorta and pulmonary artery arise side by side. A red rod is passed through the upper part of the interventricular septum into the cavity of the undeveloped left ventricle, which does not communicate with the left auricle. This auricle is very small and possesses a well-developed appendix; it has a free communication through the foramen ovale, with the dilated and hypertrophied right auricle.

Margaret B., æt. 3 months, was admitted under Dr. Hale White in a moribund condition, and died two hours later. A loud systolic bruit, associated with a thrill, was heard all over the cardiac area. At the autopsy patches of collapse were found in the lungs. There were no malformations other than those of the heart and large vessels. *See Insp.*, 1897, No. 108.

2330 Dilated Coronary Sinus.

A heart seen from behind and presenting at the situation of the coronary sinus a thin-walled sacculus measuring an inch and three quarters in its longest diameter. It

communicates by an aperture, admitting the tip of the little finger, with the right auricle. The foramen ovale is patent.

From a female infant, æt. 11 months, who died from epiphysitis and osteomyelitis. The viscera, with the exception of the heart, were normal.

Presented by MR. TARGETT, 1900.

2424 Chronic Endocarditis of Aortic Valve.

An infant's heart laid open to show the aortic cusps thickened, two of them being coherent. At the point of union of these cusps, there is a small mass of fibrinous deposit.

Ellen L., æt. 11 months, was admitted under Dr. Pitt in a moribund condition and died on the following day. The child had been in the hospital a short time previously and was then noticed to have a systolic basic bruit, which was believed to be due to congenital heart disease. Before death a to-and-fro bruit was heard at the base of the heart. At the autopsy the lungs presented patches of collapse, the other viscera being normal. See *Insp.*, 1892, No. 465; and *Trans. Path. Soc.*, vol. xlv., p. 28.

2426 Ulcerative Endocarditis of Aortic Valves.

A portion of a heart mounted to show the aortic valves extensively destroyed by ulceration and partially encrusted by fibrinous deposit. On the reverse of the specimen a similar deposit is seen attached to the wall of the pulmonary artery a little above the valve, which itself appears to be normal.

Francis S., æt. 25, was admitted under Dr. Perry with signs of valvular disease of the heart, supposed to be due to an attack of rheumatic fever, from which the patient had suffered six months previously. He died ten days after admission, and at the autopsy the heart was found to weigh nineteen ounces. There were patches of broncho-pneumonia in the lungs and the kidneys were infarcted. See *Insp.*, 1891, No. 340.

2429 Ulcerative Endocarditis. Perforation of Aortic Valve.

A portion of a heart mounted to show ulcerative endocarditis affecting the aortic and mitral valves, the aortic being also affected by chronic disease. The two anterior semilunar cusps are much thickened, and are fused so as to form a single large flap. The posterior

cusps presents a rounded perforation fringed with vegetations. In the neighbourhood of the valves are seen the orifices of three small aneurysms. The aortic cusps of the mitral valve together with its chordæ tendiniæ presents numerous small septic vegetations.

Joseph H., æt. 46, was admitted under Dr. Taylor for dyspnoea and dropsy, having previously suffered from rheumatism. On admission bruits were heard in both mitral and aortic areas and there was a petechial rash upon the legs. The patient died the day after admission, and at the autopsy the liver and spleen were found to be enlarged and the kidneys were scarred. *See Insp.*, 1894, No. 195.

2457 Aortic Stenosis.

The base of a heart mounted to show the aortic cusps forming a rough dome-like septum, with its convexity towards the arch of the aorta. The valve is extremely rigid and calcareous, and the aperture resembles a button-hole rather less than half an inch in length. There is a considerable calcareous deposit in the mitral valve continuous with that in the semilunar cusps.

From a hawker, æt. 37, who was found dead in the snow. His friends believed him to have been in good health. The heart weighed fifteen ounces.

Presented by MR. A. C. ELLIMAN, 1891.

2458 Aortic Stenosis.

The base of a heart mounted to show the aortic orifice obstructed by the cohesion of the semilunar cusps. Two of the cusps are united to form a thick fibrous septum, whilst the left anterior cusp presents a large nodular excrescence, bare of endothelium and infiltrated with calcareous deposit. The mitral valve is also thickened. The epicardium is roughened by old adhesions.

Louisa H., æt. 58, was admitted under Mr. Higgins with opacity of the cornea and closure of the left pupil, for which iridectomy was performed. There were physical signs of aortic and mitral disease, with symptoms of chronic cardiac failure, from which she died nine days after her admission. At the autopsy the heart was found to weigh thirteen ounces, and the lungs were in a condition of brown induration. *See Insp.*, 1891, No. 193.

2462 Stenosis of Cardiac Valves.

The base of a heart mounted to show a considerable degree of stenosis of the aortic, mitral, and tricuspid valves. The pulmonary valve is normal. There are recent vegetations on the mitral curtains.

Emma M., æt. 30, was admitted under Dr. Taylor for general œdema and hæmoptysis, associated with a systolic bruit at the apex of the heart. She had had four attacks of acute rheumatism. The patient died four days after admission, and at the autopsy the heart was found to weigh fifteen ounces and the viscera were congested. *See Insp.*, 1895, No. 61.

2463 Aortic Stenosis. Fibroid Heart.

A portion of a heart, which in the recent state weighed twenty-two ounces, mounted to show the aortic orifice reduced to a narrow chink, half an inch in length, between the edges of two of the cusps. The valves are thickened and loaded with calcareous deposit. A section through the hypertrophied wall of the left ventricle shows several white patches of fibroid change in the myocardium. The other valves are healthy.

George R., æt. 61, was admitted under Dr. Taylor for dyspnoea and anasarca of nine months' duration. He had had a winter cough for twenty years. On admission there were physical signs of pleuritic effusion, and of aortic stenosis and mitral regurgitation. Paracentesis thoracis was performed, and the patient died two days after his admission. At the autopsy the heart was found to weigh twenty-two ounces, the kidneys were granular, and there was a deposit of urate of soda in the great-toe joint. *See Insp.*, 1898, No. 406.

2505 Four Pulmonary Semilunar Valves.

A portion of a pulmonary artery considerably hypertrophied and measuring four inches in circumference. Its orifice is guarded by four nearly equal semilunar cusps, all of which are thickened and opaque but of normal shape.

From a congenital idiot who died at the age of 50 in the Norfolk County Asylum. He had suffered from subacute rheumatism and died from chronic cardiac failure. At the autopsy the heart was found to weigh thirty-two ounces, the mitral orifice being considerably dilated. The viscera were congested.

Presented by DR. J. ROBERTSON, 1898.

2521 Pulmonary Stenosis.

The base of a heart mounted to show the cusps of the pulmonary valve united to each other to form a thin conical septum, in the summit of which is seen a narrow opening measuring half an inch in length and a line in width. On the edge of the orifice there are a few minute vegetations.

Phœbe B., æt. 50, was admitted under Dr. Pye-Smith for an abdominal tumour associated with vomiting and emaciation. There was a loud rasping systolic bruit audible all over the chest, loudest in the second left intercostal space. The patient died about four months after admission, and at the autopsy a malignant growth was found in the colon invading the duodenum. The heart weighed nine and a half ounces. *See Insp.*, 1899, No. 10.

AORTA.

2538 Coarctation of the Aorta. Hypertrophied and Dilated Heart.

An aorta with the base of the heart. At the junction of the descending aorta with the arch there is a constriction whereby the lumen of the vessel is narrowed so as to barely admit a fine probe. Below the constriction the vessel is normal, while above it is considerably thickened by atheromatous deposit, and somewhat diminished in calibre for a distance of about an inch. The aortic valves, two in number, are thickened, and the adjacent portion of the aorta atheromatous.

Thomas M., æt. 45, was admitted under Dr. Pye-Smith for dyspnoea and cedema of the legs. He had had "rheumatic gout" seven years previously, and was in bed two months. Since this time he had suffered with cough and palpitation of the heart. In October, 1898, he was admitted under Dr Pitt with pleurisy, associated with hypertrophy of the left ventricle and albuminuria. At the autopsy the heart was found to be generally hypertrophied and dilated, weighing twenty-three ounces. The lungs were in a condition of "carnification," and there were some recent infarcts in the kidneys. *See Insp.*, 1899, No. 142.

2539 Occlusion of Aorta.

The base of the heart with the great vessels and the lower end of the trachea. At a point immediately below that at which the still patent ductus arteriosus joins the aorta the vessel is completely occluded. Below the site of the

occlusion, the aorta, which appears thin and healthy, is diminished in size by about one-half, and above as far as the origin of the left subclavian artery it is still further diminished.

Eliza S., æt. circa 45, was admitted under Dr. Horrocks for pregnancy and heart disease. She had born eight children alive, the last pregnancy being one year ago. On admission she was dyspnoëic and had some bronchitis and albuminuria, in addition to signs of valvular disease of the mitral and aortic orifices. Labour was induced, but patient's condition did not improve, and she died seventeen days after admission. At the autopsy aortic stenosis was found. *See Insp.*, 1899, No. 441.

2540 Malposition of the Aorta.

A heart with its great vessels, the trachea, and the main divisions of the bronchi dissected to show the aorta crossing the right bronchus and passing behind the lower end of the trachea. There is no innominate artery, the right common carotid and subclavian arteries arising directly from the arch of the aorta. The ductus arteriosus, which springs from the pulmonary artery in the normal position, crosses the left bronchus to be attached to the left subclavian artery.

Eliza D., æt. 7½, was admitted under Mr. Lucas after having been run over. She died as the result of the injuries received. *See Insp.*, 1888, No. 379.

2542 Aorta perforated by a Bone impacted in the Œsophagus.

A portion of a thoracic aorta and of an œsophagus, mounted to show a fistulous communication between the two tubes indicated by a red rod. The opening in the aorta is situated just below the origin of the subclavian artery: On the wall of the œsophagus opposite to the perforation is an ulcer leading into the connective tissue between the œsophagus and the trachea. In the recent state a piece of chicken-bone occupied the position of the rod.

Francis N., æt. —, was admitted under Dr. Goodhart, having swallowed a chicken-bone a week previously. On the evening before she died she was found in a fainting condition on her bed, and on being raised she immediately vomited about half a pint of blood.

Hæmorrhage recurred on two occasions during the next few hours, and eventually proved fatal. At the autopsy seven ounces of blood-clot were found in the stomach, and the intestines also contained a considerable quantity. *See Insp.*, 1898, No. 112.

2547 Rupture of the Aorta into the Pericardium.

A left ventricle with the first part of the aorta laid open to show, an inch above the semilunar valves, a ragged laceration through the inner coat of the vessel, large enough to admit the tip of the little finger. On the reverse of the specimen the outer coats of the aorta are seen to have been separated over a considerable area, and to present a longitudinal tear, which, in the recent state, communicated with the pericardial sac. The interior of the aorta appears healthy. At the apex of the left ventricle there are some patches of fibroid myocarditis, over which the pericardium has been adherent.

From a woman, æt. 26, who was found dead in front of a grate that she was about to clean, having been seen in her usual health a few minutes before. She had been confined of a seven-months' child at twenty-one years of age, and had suffered from symptoms of syphilis. At the autopsy the pericardium was found distended with about a pint and a half of fluid blood, in addition to a large amount of coagulum.

Presented by DR. J. COLLISON MORLEY, 1889.

2550 Aortitis.

The first part of an aorta laid open to shew the vessel, for a distance of two inches from the semilunar valves, to be thickened and its interior irregularly corrugated. This condition is clearly defined from the healthy aorta beyond by a raised sinuous margin. The semilunar valves are thickened and much deformed, and appear to have been partially destroyed by ulceration.

Mary S., æt. 14, was admitted under Dr. Taylor for anæmia and dyspnœa, with an enlarged heart and aortic incompetence. She died shortly after an attack of vomiting. At the autopsy the heart was found to be generally dilated and hypertrophied. The mitral, tricuspid, and pulmonary valves were all normal. There were infarcts in the lungs and kidneys. *See Insp.*, 1901, No. 332.

The base of a heart with the first part of the aorta. The semilunar valves are greatly thickened and deformed. Two are united together, their point of union being marked by a mass of calcareous deposit. Just above the valves there is a localised thickening of the aorta, forming a raised plaque with a well-defined sinuous margin which almost encircles the vessel and extends upwards for a distance of one inch.

John S., a blacksmith, æt. 31, was admitted under Dr. Pye-Smith for pain in the chest, dyspnœa, and œdema of the legs, associated with an enlarged heart and a "to-and-fro," bruit, which was audible all over the front of the chest. Death occurred suddenly, fourteen days later. At the autopsy the heart was found to be dilated and hypertrophied (weight twenty-two ozs.), and on the surface of the liver were several scar-like patches, which, however, did not penetrate the substance of the organ. The testes were healthy. *See Insp.*, 1896, No. 7.

2564 Aneurysm of the Aorta and Innominate Artery surrounding the Clavicle.

A portion of the arch of an aorta, which is extremely atheromatous, and has been laid open to shew at its upper part the orifice of an aneurysmal sac plugged by firm blood-clot. Above the aorta is seen the sac of the aneurysm which measures about four inches in diameter. Its wall has been partially removed, and reveals the greater part of the right clavicle, which, bared of its periosteum and eroded, has been separated from its sternal end and projects into the sac. A red rod marks the course of the arch of the aorta.

Alfred H., æt. 62, was admitted under Dr. Fawcett for a large aneurysm of gradual development projecting from the upper part of the right side of the chest. There was a history of venereal disease. At the autopsy the right pleura was found to be thickened and adherent and the right lung compressed, as was also the trachea to a slight degree. Parts of the manubrium, the clavicle, and the first two ribs were eroded. The outer wall of the aneurysm was formed by some of the muscles attached to the clavicle and the overlying skin. The aorta was dilated, and very atheromatous throughout. *See Insp.*, 1902, No. 30.

2575 Aneurysm of the descending Aorta communicating with the Œsophagus and the Left Bronchus.

Portions of the thoracic aorta and of the œsophagus and trachea. The aorta is extremely atheromatous and dilated, and in the recent state presented a saccular aneurysm arising from the descending portion, and eroding the bodies of some of the dorsal vertebræ. The red rod marks an opening from the wall of the aneurysm into the adjacent cellular tissue. There are openings in the œsophagus and the left bronchus, whereby an indirect communication was established between these tubes and the perforation in the wall of the aneurysm.

John G., æt. 53, was admitted under Sir Cooper Perry for severe pain in the chest, of some months' duration, increased by taking food. Death took place suddenly from hæmorrhage. At the autopsy the trachea and main bronchi were filled with recent clot. *See Insp.*, 1904, No. 30.

2577 Aneurysm of the Aorta opening into the Pericardium.

The base of a heart with the first part of the aorta laid open to shew, two inches above the semilunar valves, the circular orifice of an aneurysmal sac, which admits the thumb. The sac is ovoid in shape, measuring four inches in its longest diameter, and presents on its lower and anterior aspect a small laceration into the pericardial cavity.

Frank F., æt. 24, was admitted under Dr. Shaw, in a collapsed condition, having fainted while at work an hour previously. He was almost pulseless, and his lips and face were blanched; he died five minutes after reaching the ward. At the autopsy a gumma was found occupying the apex of the left ventricle, and another in the pancreas, *See Insp.*, 1899, No. 151.

2578 Aneurysm of the Aorta opening into the Pericardium.

The base of a heart with the first part of the aorta laid open to shew, just above the junction of the anterior and right posterior cusps, the circular orifice of an aneurysmal sac, through which the tips of three fingers can be passed.

The sac is ovoid in shape, has a thin wall, and measured three and a half inches in its longest diameter. In the recent state, a small communication was discovered between the sac and the pericardial cavity. The surface of the pericardium is roughened by a soft fibrinous deposit.

Arthur P., æt. 30, was admitted under Dr. Washbourn, having been found in his cart in a moribund condition. He was cyanosed and in intense pain. There was a great increase in the area of præcordial dulness. Subsequently pulsation was detected to the right of the sternum and a pericardial rub was heard. On the morning of his death he had an attack of syncope, and succumbed in a few minutes. At the autopsy the pericardium was found to contain sixteen ounces of blood-clot, some of which was decolorised. *See Insp.*, 1896, No. 179.


2590 Aneurysm of the Arch of the Aorta invading the Trachea.

The arch of an aorta laid open to shew on its posterior wall, immediately beneath the origin of the common carotid and subclavian arteries, a smooth circular opening large enough to admit the tip of the little finger, and leading into an aneurysmal cavity about the size of a walnut. On the reverse of the specimen the sac is seen to have bulged into the trachea, considerably narrowing its lumen. On the prominence thus produced there are two teat-like projections, over which the mucous membrane is partially destroyed, exposing the blood-clot in the sac.

Albert D., æt. 42, was admitted under Dr. Taylor for a cough of about six weeks' duration, during which period he had on one occasion brought up about an egg-cupful of bright red blood. He was very dyspnoeic, and a loud respiratory stridor was present. Signs of bronchitis were audible all over the lungs, and he shortly developed pneumonia, to which he succumbed. At the autopsy diffuse broncho-pneumonia with pleurisy was found, and the aorta was atheromatous. *See Insp.*, 1904, No. 142.

2592 Aneurysm of the Aorta compressing the Superior Vena Cava.

Half of the ovoid sac of an aortic aneurysm, measuring five inches from above downwards and three inches from side to side. It arises from the first part of the aorta, and the aortic orifice, guarded by its semilunar valves, can be



seen on the middle of the lateral wall of the sac. The superior vena cava, which courses over the posterior wall of the sac, has been laid open to shew its lumen considerably encroached upon by the pressure of the aneurysm.

Henry J. T., æt. 50, was admitted under Dr. Shaw for orthopnœa cyanosis and dulness over the right side of the chest. There was some bulging of the chest wall, but no pulsation. General anasarca was present. At the autopsy, parts of the sternum and of the third, fourth and fifth ribs were eroded. The right pleura was thick and adherent, and there was double hydrothorax. *See Insp.*, 1890, No. 28.

2600 Aorta opened by Epitheliomatous Ulcer of the Œsophagus.

A thoracic aorta, with the œsophagus laid open from behind, to shew a small oval communication between the two tubes situated about two inches below the level of the bifurcation of the trachea. In the aorta the edges of the perforation are smooth and free from evidence of disease, while in the œsophagus there is a crateriform malignant ulcer, the base of which is partly formed by the outer wall of the aorta. Histologically the wall of the ulcer is infiltrated with squamous-celled epithelioma.

William S., æt. 52, was admitted under Dr. Taylor for difficulty in swallowing, and died suddenly, six weeks later, from profuse hæmorrhage. At the autopsy the stomach was found to be distended by a large mass of blood-clot. *See Insp.*, 1895, No. 106.

2601 Aorta opened by Epithelioma of the Œsophagus.

A thoracic aorta mounted to shew, at the junction of the arch with the descending portion, a small opening communicating with the œsophagus. This opening is seen to be situated in the midst of a large cancerous ulcer, which involves the upper two-thirds of the gullet, and histologically has the structure of a squamous epithelioma.

From a man who died suddenly after bringing up a large quantity of blood.

Presented by DR. BRYAN, of Leicester Infirmary, 1898.

A portion of the thoracic aorta, presenting in its course a sharp bend with its concavity to the right, which resulted from a curvature of the spine, due to caries. The aorta is very small, and its inner coat is somewhat atheromatous.

Alice M., æt. 33, was admitted under Dr. Pye-Smith for symptoms of lardaceous disease following upon psoas abscess and spinal caries. She died six days after admission, and at the autopsy the spine was found to be affected by caries in the dorsal and lumbar regions. There was interstitial and tubal nephritis. *See Insp.*, 1897, No. 278.

2608 Calcification of the Abdominal Aorta.

An abdominal aorta with its terminal branches laid open from behind to shew its lining to be thickened and irregular, and in some places removed by ulceration. The deeper coats are infiltrated by calcareous deposit, which for the last two inches of the aorta has converted the vessel into a rigid bony tube.


Joseph H., æt. 72, was admitted under Mr. Golding Bird for gangrene of the right foot. The leg was amputated, but two days later the stump became gangrenous, and he died four days afterwards from asthenia. At the autopsy the heart was found to weigh ten ounces, and the valves were competent. There was very little atheroma in the ascending portion and arch of the aorta, but the iliac arteries and those of the lower extremities shewed considerable calcareous degeneration of their coats. *See Insp.*, 1895, No. 465.

2617 Healed Aneurysm of the Abdominal Aorta.

A portion of the abdominal aorta with its terminal branches laid open from behind to shew, just above its bifurcation, an aneurysmal sac, produced by the protrusion of the anterior wall of the vessel over a distance of three inches. The wall is rigid from calcareous deposit, and the interior is partially filled with firm blood-clot.

This specimen was taken from a woman who died in the Stepney and Poplar Sick Asylum in 1897.

Presented by MR. A. B. CARTER.



2624 Embolism of the Abdominal Aorta.

The lower end of an aorta with its terminal branches, as far as the femoral arteries. There is a large mass of firm partially decolorised blood-clot occupying the last inch of the aorta, and occluding both common iliac arteries. From this mass of thrombus there extends softer and dark red blood-clot into the greater number of the vessels forming the preparation.

Emma H., æt. 40, was admitted under Dr. Perry in a collapsed condition, with an irregular and very rapid pulse, and died twenty-one hours later. She had been seized with acute pain in the abdomen, followed by sickness, eight hours before admission. Albumen was present in the urine. At the autopsy an extreme condition of mitral stenosis was found, and the left auricular appendix was full of antemortem clot, which projected out into the cavity of the auricle. The left renal artery was plugged by clot; the aorta was free from disease. *See Insp.*, 1903, No. 15.

PULMONARY ARTERY.

2628 Atheroma of the Pulmonary Artery

A heart with the pulmonary artery laid open to shew numerous patches of atheroma situated chiefly in the main divisions of the vessel, the trunk itself appearing normal. The patches are raised, and of a yellowish colour, contrasting with the healthy surrounding parts. The right ventricle is dilated and hypertrophied, its wall measuring half an inch in thickness; the tricuspid valve is thickened and the mitral orifice considerably narrowed.

Henry W., æt. 22, was admitted under Dr. Taylor, for heart disease, for which he had been an in-patient three times previously during 1891-1892. The physical signs pointed to the presence of mitral stenosis and regurgitation, with tricuspid incompetence. At the autopsy the heart was found to weigh eighteen ounces. The branches of the pulmonary artery in the lungs were dilated and atheromatous. *See Insp.*, 1894, No. 81.

2637 Thrombosis of the Pulmonary Artery.

A pulmonary artery with its main divisions laid open to shew, in its right branch, a mass of adherent thrombus,

having a granular surface and a pointed extremity towards the heart.

John N., æt. 20, was admitted under Dr. Taylor for morbus cordis, from symptoms of which he had suffered for six years. He presented the physical signs of mitral stenosis and tricuspid incompetence with general anasarca. While in the hospital he had numerous attacks of urgent dyspnœa, in one of which he died. At the autopsy the right side of the heart was found to be enlarged, both mitral and tricuspid valves being fibrous and shrunken. There was thrombosis in the right ventricle and the left subclavian vein, and in the lower part of the inferior cava extending into each iliac vein. There was old and recent infarction of lungs and kidneys. *See Insp.*, 1892, No. 237.

2642 Sarcomatous Thrombus in Pulmonary Artery.

The pulmonary artery of a child, mounted, to shew in its left division a mass of malignant deposit adherent to its wall and partially occluding its lumen. The deposit is of soft consistency, has a rough surface, and histologically has the structure of a sarcoma.

Thomas M., æt. 5, was admitted under Mr. Durham for a large tumour on the left side of the abdomen, which proved to be a sarcoma of the kidney. The child became emaciated and dyspnœic, and during the latter part of his life cyanosed. At the autopsy the liver was found to be much enlarged by secondary deposits. The kidney-tumour weighed thirty-four ounces. *See Insp.*, 1893, No. 11; and *Trans. Path. Soc.*, vol. xliv., 1893, p. 48.

LIST
OF
GENTLEMEN EDUCATED AT GUY'S HOSPITAL
WHO HAVE PASSED THE
EXAMINATIONS OF THE SEVERAL UNIVERSITIES, COLLEGES,
&c., &c.,
IN THE YEAR 1903.

University of Oxford.

Degree of Doctor of Medicine.

G. G. Davidson.

Degree of Master in Surgery (M.Ch.)

F. E. Fremantle.

Second M.B. Examination.

Medicine, Surgery, Midwifery, Forensic Medicine and Public Health.

J. M. Bickerton.	P. N. Blake Odgers.	W. E. Robinson.
	A. R. Wilson.	

University of Cambridge.

Degree of Doctor of Medicine.

J. G. Taylor.

Final Examination for the Medical and Surgical Degrees.

Part II.

H. A. Ackroyd.	J. S. Cooper.	C. M. Murray.
E. Bigg.	H. A. Cutler.	F. Richmond.
A. R. Brailey.	J. H. Donnell.	R. D. Smedley.
W. H. Brailey.	F. B. Manser.	A. Wylie.

Part I.

A. R. Brailey.	R. E. French.	C. W. Ponder.
S. Child.	A. H. Miller.	C. M. Stevenson.
J. S. Cooper.	F. W. M. Palmer.	B. H. Stewart.
	A. Wylie.	

402 *Gentlemen admitted to Degrees, &c., in the year 1903.*

Second Examination for the Medical and Surgical Degrees.

F. A. Barker.	C. F. Fothergill.	C. W. Greene.
R. Davies-Colley.	G. W. Goodhart.	G. W. de P. Nicholson.

Examination in Sanitary Science.

A. Armer.	G. E. Richmond.	G. Warwick Smith.
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University of London.

Examination for the Degree of Doctor of Medicine.

T. P. Berry.	F. G. Gibson.	W. M. Robson.
G. Clarke.	S. Hodgson.	L. E. Stamm.
D. Forsyth.	G. E. Richmond.	C. Tessier.

H. Nolan (State Medicine).

Obtained the Gold Medal.

Examination for the Degree of Master in Surgery.

G. E. Manning.	R. P. Rowlands.
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Examination for the Degree of Bachelor of Surgery.

First Division.

H. Watts.

Obtained the Gold Medal.

C. H. Robertson.

Obtained Honours.

H. McD. Parrott.	A. M. Webber.	G. T. Wrench.
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Second Division.

J. Evans.	G. Lewin.	N. Ivens Spriggs.
T. Holmes.	E. H. B. Milsom.	W. C. Swayne.

Examination for the Degree of Bachelor of Medicine.

May.

Second Division.

W. F. Box.	E. G. Goldie.	H. McD. Parrott.
J. Braithwaite.	A. C. H. Gray.	G. W. Smith.
H. S. Brown.	G. Lewin.	E. W. Strange.
F. W. Fawcett.	L. H. Moiser.	H. Tipping.
L. S. H. Glanville.	D.L. Morgan.	D. H. Trail.
		A. H. E. Wall.

October.

First Division.

G. T. Wrench.

Obtained Honours in Medicine and Forensic Medicine.

C. D. Pye-Smith.

H. Watts.

Second Division.

N. Ivens Spriggs.

Obtained Honours in Medicine and Forensic Medicine.

A. M. Webber.

Obtained Honours in Forensic Medicine.

P. R. Bolus.

R. A. Greeves.

E. H. B. Milsom.

H. M. Goldstein.

R. Larkin.

H. F. B. Walker.

B. H. Wedd.

Intermediate Examination in Medicine.

January.

Entire Examination.

Second Division.

E. H. Adams.

E. M. Harrison.

W. P. Purdom.

T. H. Barton.

H. S. Knight.

F. A. Sharpe.

R. J. Bentley.

E. F. Milton.

H. A. Watney.

G. Hamilton.

T. C. Pocock.

F. T. H. Wood.

Physiology only.

Second Division.

I. R. Cook.

July.

Honours Examination.

G. Cockcroft.

Obtained the Exhibition and Medal in Anatomy and Honours in Physiology and Histology.

T. B. Layton.

Obtained First Class Honours in Anatomy.

J. S. Bookless.

Obtained Honours in Anatomy.

Entire Examination.

First Division.

W. H. Trethowan.

Second Division.

E. Alban.

E. C. Lowe.

P. S. Mills.

A. W. Berry.

P. F. McEvedy.

H. F. Vandermin.

C. C. A. De Villiers.

W. H. Miller.

T. F. Wilson.

Physiology only.

Second Division.

P. C. P. Ingram.

Preliminary Scientific (M.B.) Examination.

January.

Entire Examination.

First Division.

S. C. H. Air.		E. L. W. Mandel.		M. D. Price.
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Second Division.

H. R. Bastard.		A. L. Gardner.		H. E. Perkins.
R. C. V. Edsall.		J. B. Martin.		S. G. Tracy.

Chemistry and Experimental Physics.

M. E. Ball.		T. Evans.		B. McDermott.
A. F. W. Denning.		H. J. Henderson.		T. E. Price.
J. B. Dunning.		K. H. Hole.		G. F. Syms.

Biology.

S. S. Brook.		G. B. Harland.		D. Reynolds.
		H. J. Smith.		

July.

Entire Examination.

First Division

M. M. Adams.		H. B. Carter.		E. L. M. Lobb.
		T. Stansfield.		

Second Division.

S. Chelliah.		W. Johnson.		A. E. Lees.
		P. S. Price.		

Chemistry and Experimental Physics.

S. S. Brook.		G. B. Harland.
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Biology.

M. E. Ball.		K. H. Hole.		B. McDermott.
J. B. Dunning.		H. C. Lucey.		N. A. D. Sharp.

Intermediate Examination in Science.

Honours Candidate recommended for a Pass.

S. G. Tracy.

Intermediate Examination in Science and Preliminary Scientific Examination conjointly.

H. A. Sanford.

University of Durham.

Examination for the Degree of Doctor in Medicine.

H. C. Sturdy.

*Examination for the Degree of Doctor of Medicine for Practitioners
of Fifteen Years' Standing.*

G. F. Hugill.

E. Sharpley.

*Final Examination for the Degrees of Bachelor of Medicine
and Surgery.*

B. Glendining.

J. G. O. H. Lane.
A. Reid.

A. A. Miller.

Third Examination.

C. M. Anthony.

B. W. Lacy.

Chemistry and Physics.

First Examination.

L. K. Edmeades.

J. F. Young.

T. H. V. King.

Anatomy and Biology.

H. C. W. Allott.

H. F. Joynt.

E. R. H. Joynt.

Royal College of Physicians of London.

Elected to the Fellowship.

A. P. Beldard.

E. Goodall.

Examination for the Diploma of Membership.

J. G. Taylor.

Final Examination for the License.

January.

W. F. Box.
A. R. Brailey,
S. Child.

W. H. Cole.
F. W. Fawcett.
P. W. Hamond.

A. W. Iredell.
C. B. Penny.
V. M. Wallis.

April.

J. D. Bridger.	H. C. C. Mann.	F. C. Robinson.
G. L. Buckeridge.	G. Moir.	C. R. Shattock.
C. H. Dawe.	J. D. Pearson.	H. Watts.
J. F. Douse.	B. I. Rahim.	B. H. Wedd.
E. G. Goldie.	W. W. Read.	H. C. Winckworth.
H. M. Goldstein.	E. Roberts.	

July.

K. Black.	C. E. Iredell.	C. H. Reinhold.
P. R. Bolus.	R. Larkin.	H. D. Smart.
J. S. Cooper.	E. H. B. Milsom.	N. I. Spriggs.
C. F. Fraser.	P. A. Peall.	F. L. Thomas.
J. Goss.	A. P. Piggott.	
G. W. C. Hollist.	C. D. Pye-Smith.	

October.

R. G. Anderson.	H. S. Jones.	A. M. Webber.
P. C. V. Bent.	W. Collins Lewis.	R. Willan.
S. C. Bowle.	F. C. Lucas.	
W. Johnson.	F. H. Wallace.	

Royal College of Surgeons of England.

Final Examination for the Fellowship.

J. H. Atkins.	F. E. Fremantle	E. A. Peters.
W. H. Bowen.	J. G. O. H. Lane.	C. H. Robertson.
P. W. L. Camps.	W. G. Mumford.	

First Examination for the Fellowship.

J. A. Andrews.	T. H. Barton.	A. G. Jones.
J. B. Ball.	L. H. Burner.	T. B. Layton.
A. S. Bankart.	G. Cockcroft.	G. C. F. Robinson.
G. N. Bartlett.	R. Davies-Colley.	W. Welchman.

Examination for the Diploma in Public Health.

R. F. Clark.	H. Hewetson.
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Final Examination for the Membership.

January.

W. F. Box.	W. H. Cole.	A. W. Iredell.
A. R. Brailey.	F. W. Fawcett.	C. B. Penny.
S. Child.	P. W. Hamond	V. M. Wallis.

April.

J. D. Bridger.	H. C. C. Mann.	F. C. Robinson.
G. L. Buckeridge.	G. Moir.	C. R. Shattock.
C. H. Dawe.	J. D. Pearson.	H. Watts.
J. F. Douse.	B. I. Rahim.	B. H. Wedd.
E. G. Goldie.	W. W. Read.	H. C. Winckworth.
H. M. Goldstein.	E. Roberts.	

July.

K. Black.	C. E. Iredell.	C. H. Reinhold.
P. R. Bolus.	R. Larkin.	H. D. Smart.
J. S. Cooper.	E. H. B. Milsom.	N. I. Spriggs.
C. F. Fraser.	P. A. Peall.	F. L. Thomas.
J. Goss.	A. P. Piggott.	
G. W. C. Hollist	C. D. Pye-Smith.	

October.

R. G. Anderson.	H. S. Jones.	A. M. Webber.
P. C. V. Bent.	W. Collins Lucas	R. Willan.
S. C. Bowle.	T. C. Lucas.	
W. Johnson.	F. H. Wallace.	

Society of Apothecaries of London.

C. E. Adams.	E. H. Griffin.	F. J. Turner.
J. E. L. Bates.	D. R. T. Griffith.	A. J. Urquhart.
J. C. V. Bradbury.	G. R. S. Soper.	
H. N. Collier.	W. A. G. Stevens.	

Naval Medical Service.

G. L. Buckeridge.	A. W. Iredell.	F. M. V. Smith.
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Royal Army Medical Corps.

H. V. Bagshawe.	F. M. M. Ommanney.	G. Warwick Smith.
R. T. Collins.	S. L. Pallant.	F. J. Turner.
A. C. H. Gray.	C. H. Robertson.	M. C. Wetherell.

Indian Medical Service.

H. C. Keats.	W. E. J. Tuohy.
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MEDALLISTS AND PRIZEMEN,

JULY, 1904.

Open Scholarships in Arts.

Sydney John Darke, Owen's School, Islington, £100.
 Jean René Perdrau, Royal College, Mauritius, £50.
 Guy Barton Cockrem, King's School, Canterbury, Certificate.

Open Scholarships in Science.

Harry Archibald Sanford, Guy's Hospital, £150.
 Leonard Tilsley Baker, Dulwich College, £60.
 William Johnson, Guy's Hospital
 Vincent Townrow, University College, Sheffield } Equal
 Certificates.

Scholarship for University Students.

Charles William Greene, Emmanuel College, Cambridge, £50.

Open Scholarships in Dental Mechanics.

October, 1903, Robert Charles Basker Shaw, £20.
 May, 1904, George Packham, £20.

Junior Proficiency Prizes.

Kenelm Hutchinson Digby, £20.
 Ernest William Giesen, £15.
 Thomas Edmund Ashdown Carr, £10.
 George French Stebbing, Certificate.

The Michael Harris Prize for Anatomy.

Kenelm Hutchinson Digby, £10.

The Sands Cox Scholarship in Physiology.

Ernest William Giesen, £15.
 Kenelm Hutchinson Digby, Certificate.

The Hilton Prize for Dissections (1903).

Patrick Francis McEvedy, £3.
 William Henry Miller, £1.
 William Henry Trethowan, £1.
 (1904) Kenelm Hutchinson Digby, £1 13s. 4d.
 Ernest William Giesen, £1 13s. 4d.
 Christian Hugo Rippman, £1 13s. 4d.

The Arthur Durham Prizes for Dissection.

First Year's Students.

Edward Leslie Martyn Lobb, £5.

Thomas Stansfield, Certificate.

William Johnson, Certificate.

Howard Bernard Carter, Certificate.

Senior Students.

Ernest William Giesen, £15.

Dental Prizes.

First Year's Students.

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Arthur William Parrott, Certificate.

Practical Dentistry Prize.

James William Mawer, £10.

Julian Bollard, Certificate.

Travelling Dental Scholarship.

John Edmund Spiller, £100

The Beaney Prize for Pathology (1903).

Neville Ivens Spriggs, £34.

The Treasurer's Gold Medal for Clinical Medicine.

Arthur Frederick Hertz.

The Treasurer's Gold Medal for Clinical Surgery.

Arthur Frederick Hertz.

The Golding-Bird Gold Medal and Scholarship in Bacteriology.

Charles Morley Wenyon, £20.

Maurice George Louisson, Certificate.

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The Society's Prize of £10 was awarded to Mr. N. Ivens Spriggs, M.B., B.S., for his paper on "The Diagnosis and Treatment of Coma," and Mr. J. Sephton Cooper, M.A., M.B., B.C., gained the Treasurer's Prize of £5 for his paper, "The Uses of Light and Electricity in Surgery."

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G. T. Wrench.	M. J. Rees.	H. A. Cutler.
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B. Glendining.	T. Morland Smith.	B. H. Wedd.
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A. R. Brailey.	H. S. Brown.	L. S. H. Glanville.

ASSISTANT HOUSE PHYSICIANS.

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H. A. Cutler.	G. Evans.	D. H. Trail.
B. Glendining.	G. E. Malcomson,	

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N. Blake Odgers.	F. D. S. Jackson	

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F. W. Fawssett.	L. S. H. Glanville.	W. F. Box.
E. W. Strange.	C. H. Reinhold.	F. H. Wallace.
G. Goldie.	P. A. Peall.	B. Glendining.
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G. A. Ticehurst.	C. D. Pye-Smith	A. M. Webber.
H. H. Jenkins.	M. G. Louisson.	H. M. Woodward.
E. C. Myott.		

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J. Cook.		

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E. C. Myott.	E. W. Routley.	W. N. May.
J. E. Prentis.	J. E. Spiller.	S. M. Wells.
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F. Alcock.	P. F. Minett.	L. Myer.
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J. F. Minett.	L. Myer.	E. C. Hughes.
A. S. Littlejohns.	A. Leeming.	J. O. Musson.
C. M. Stevenson.	E. W. Sheaf.	R. Felton.
A. M. Roome.	H. D. Wyatt.	R. Edridge.
S. M. Wells.	P. A. S. Dyson.	F. M. Longson.
J. Cook.	H. M. Clarke.	E. White.
A. B. Cocker.	R. P. Lewis.	W. Welchman.
R. O. Williams.	H. V. Mitchell.	J. E. Scales.
M. Maher.	A. H. Clough.	L. Doudney.
C. M. Wenyon.	R. A. Chisholm.	L. G. Davies.
H. P. Costobadie.	T. Turner.	R. W. Allen.
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K. Black.	H. Watts.	E. L. Ward.
A. M. Webber.	R. G. Anderson.	M. G. Louissou.
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R. Edridge.	S. M. Wells.	A. S. Littlejohns.
C. M. Stevenson.	L. J. Orpen.	E. C. Hughes.
H. M. Clarke.	E. W. Sheaf.	N. H. Oliver.
L. Doudney.	R. O. Williams.	H. P. Costobadie.
F. M. Longson.	T. Turner.	F. H. Lennox Jones.
A. H. Clough.	A. B. Cocker.	G. F. Greening.
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M. de L. Robinson.	R. D. Barron.	H. C. Cameron.
F. D. Crew.	R. E. French.	G. H. K. Macalister.
A. H. Miller.	M. Maher.	M. McPherson.
E. Patterson-Clavier.	G. Hamilton.	W. P. Purdom.
E. H. Adams.	R. T. Bentley.	H. S. Knight.
M. Leckie.	E. F. Milton.	F. A. Sharpe.
A. H. Watney.	F. T. H. Wood.	E. B. Smith.
B. B. Metcalfe.	T. Norman.	H. T. Wight.
R. S. Harper.	E. E. Rendle.	G. W. Nicholson.
A. Morris.	T. C. Pocock.	R. M. Wingent.
G. N. Bartlett.	T. H. Barton.	A. G. Jones.
L. H. Burner.	W. S. H. Burney.	I. R. Cook.
T. R. Harvey.	E. White.	P. C. Litchfield.
J. H. Mayston.	G. H. Morris.	C. F. Fothergill.
H. G. Gibson.	A. S. M. Palmer.	

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R. P. Lewis.	T. Turner.	F. Alcock.
F. M. Longson.	W. C. Dickey.	M. de L. Robinson.
F. D. Crew.	A. H. Miller.	H. M. Langdale.
E. Patterson-Clavier.	H. C. Cameron.	G. H. Macalister.
T. C. Pocock.	F. T. H. Wood.	F. A. Sharpe.
H. F. Wight.	R. S. Harper.	E. E. Rendle.
E. B. Smith.	G. Hamilton.	

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F. D. Crew.	R. E. French.	G. W. Nicholson.
G. H. H. Macalister.	A. H. Miller.	M. Maher.
M. C. Dickey.	R. M. Barron.	M. Leckie.
R. M. Wingent.	G. Hamilton.	H. S. Knight.
E. H. Adams.	R. J. Bentley.	F. T. H. Wood.
E. F. Milton.	F. A. Sharpe.	R. S. Harper.
T. Norman.	H. F. Wight.	T. C. Pocock.
B. B. Metcalfe.	W. P. Purdom.	J. B. Ball.
H. A. Watney.	A. G. Jones.	T. R. Harvey.
L. H. Burner.	W. H. Burney.	C. F. Fothergill.
J. H. Mayston.	G. H. Morris.	A. S. M. Palmer.
H. G. Gibson.	W. H. Robinson.	I. R. Cook.
T. H. Barton.	G. N. Bartlett.	E. Morgan.
H. H. Mcyle.	P. C. Litchfield.	E. Alban.
E. M. Harrison.	E. C. Lowe.	P. F. McEvedy.
A. W. Berry.	A. W. Eyles.	C. C. De Villiers.
W. H. Miller.	E. L. R. Norton.	E. M. Ockwell.
T. F. Wilson.	P. S. Mills.	W. H. Trethowan.
S. Reader.	G. G. Timpson.	H. Vandermin.
G. Wachter.	A. Walker.	
J. S. Bookless.	C. W. R. Preston.	

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A. C. Osburn.	E. Lloyd.	E. Morgan.
G. F. Hardy.	F. P. Hughes.	

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J. Bromley.	F. H. Wallace.	H. Watts.
M. J. Mottram.	W. T. Meade King.	C. M. Murray.
B. Moiser.	F. G. Goble.	

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A. S. M. Palmer.	H. F. Wight.	F. T. H. Wood.
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H. A. Watney.	H. G. Gibson.	A. S. M. Palmer.
J. H. Mayston.	W. H. Robinson.	C. F. Fothergill.
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W. Reeve.	E. H. B. Milsom.	B. H. Stewart.
F. H. Frankenberg.	F. G. Goble.	E. Lloyd.
E. L. Ward.	E. C. Myott.	F. P. Hughes.
F. W. M. Palmer.	R. M. Rendall.	W. N. May.
H. M. Mollison.		

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A. R. Wilson.	G. S. Robertson.	J. Goss.
F. P. Hughes.	R. Larkin.	M. J. Mottram.
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L. H. Frankenberg.	J. E. Prentis.	P. P. Cole.
A. E. F. Kynaston.	C. J. S. Dismorr.	O. V. Payne.
J. McF. Pollard.	F. B. Lowe.	A. M. Benett.
A. V. Maybury.	E. J. Crew.	

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B. W. Lacy.	G. F. Hardy.	M. B. Taylor.
H. M. Woodward.	H. O. M. Beadnell.	L. H. Frankenberg.
K. Black.	F. P. Hughes.	R. A. Greeves.
R. St. G. Seagrove.	B. H. Stewart.	J. F. Rey.
P. A. Peall.	F. Barnes.	F. Rogerson.
C. S. Morris.	C. D. Pye-Smith.	A. F. Hertz.
J. F. Spiller.	A. M. Webber.	M. G. Louissou.
M. J. Mottram.	O. V. Payne.	G. A. Ticehurst.
E. Lloyd.	H. H. Carter.	W. S. Orton.
R. M. Rendall.	H. H. Jenkins.	P. P. Cole.
J. Cook.	J. D. Thomas.	H. F. B. Walker.
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D. Isaacs.	E. W. Routley.	M. Maher.
A. V. Maybury.	R. Franklin.	C. P. Harvey.
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A. R. Beaumont.	G. S. F. Robinson.	L. Myer.
J. E. Prentis.	A. M. Roome.	J. O. Musson.
J. H. Clatworthy.		

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C. F. Fraser.	B. H. Stewart.	F. G. Goble.
H. O. M. Beadnell.	R. A. Greeves.	D. Isaacs.
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J. Bromley.	W. H. Bush.	E. W. Routley.
A. V. Maybury.	E. C. Myott.	H. H. Carter.
G. A. Ticehurst.	E. C. Hughes.	R. D. Bridger.
W. M. Mollison.	A. M. Benett.	W. N. May.
G. H. Rees.	B. W. Lacy.	P. P. Cole.
F. Barnes.		

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F. W. Parfitt.		

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B. H. Martin.	N. McL. Nibbs.	

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N. McL. Nibbs.	E. L. Pilbeam.	H. Poyton.
P. H. Hickman.	J. W. Mawer.	A. Hammond Smith.

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S. H. Barlow.	V. S. Houchin.	J. F. Ryder.
R. A. Scott.	J. H. Williams.	E. White.
E. L. Pilbeam.	A. E. D. Prideaux	B. H. Martin.
R. Roberts.	H. C. Malleson.	W. H. Yeo.
F. J. Goodman.	H. Poyton.	A. Hammond Smith.
C. W. Randall.	C. L. D. Taylor.	G. E. Wood.
R. J. Messent.	H. E. H. Tracy.	H. E. Warren Williams.
J. W. Mawer.	G. G. Timpson.	R. F. Mungal.
J. B. Ball.	F. H. Fuller.	C. D. Wallis.
F. S. Vine.	H. A. Pallant.	J. H. Williams.
P. E. Luce.	F. A. Beckley.	A. H. Harris.
F. L. Aubrey.	F. A. Husbands.	H. J. Snowden.
S. D. Marshallsay.	W. Elwood.	J. Bollard.
A. A. Forty.	W. J. Wormald.	H. M. Peacock.

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H. Poyton.	T. L. Smith	G. E. Rice.
J. H. Williams.	P. H. Hickman.	S. D. Marshallsay.
R. M. Pearson.	J. S. Vogwell.	F. J. Goodman.
A. A. Forty.	F. H. Fuller.	F. A. Beckley.
H. J. Snowden.	A. Hammond Smith.	E. J. Sheppard.
P. E. Luce.	J. W. Mawer.	F. S. Vine.
W. Elwood.	W. J. Wormald.	F. A. Husbands.
W. J. C. Timberlake.	J. Bollard.	G. E. Wood.
F. L. Aubrey.	H. J. Russell.	H. G. Pearce.
W. E. Cook.	H. A. Pallant.	L. B. Moore.
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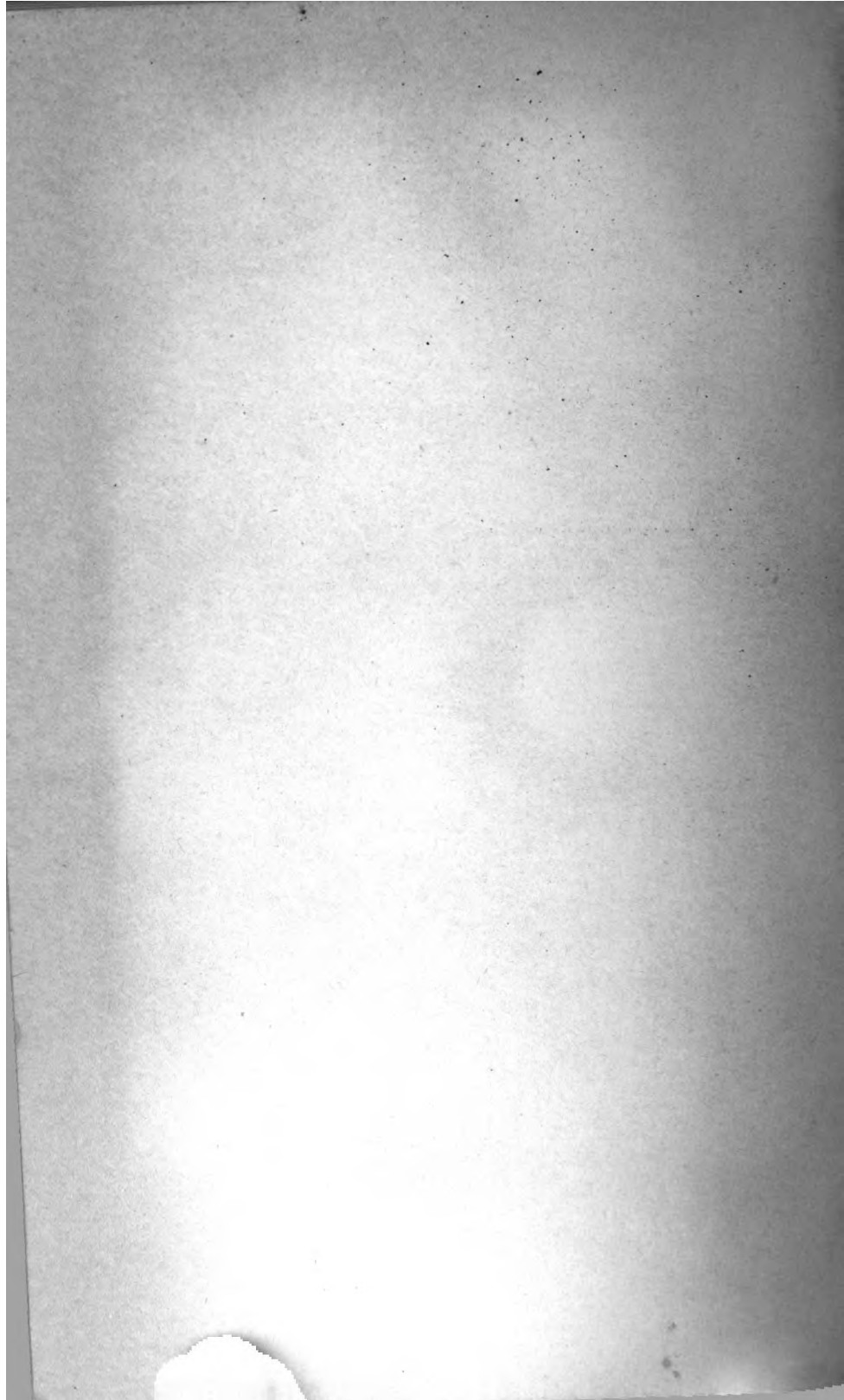
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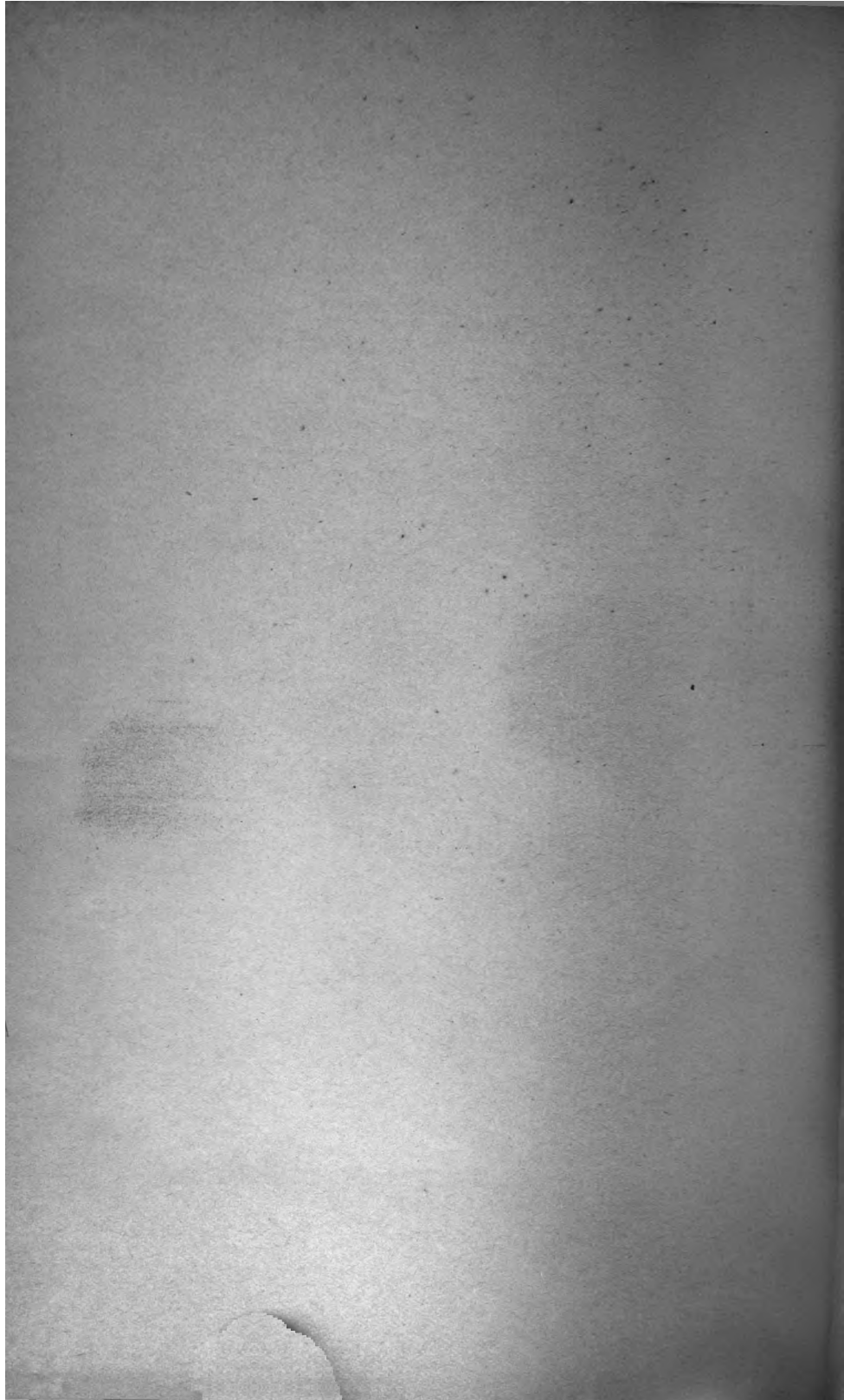
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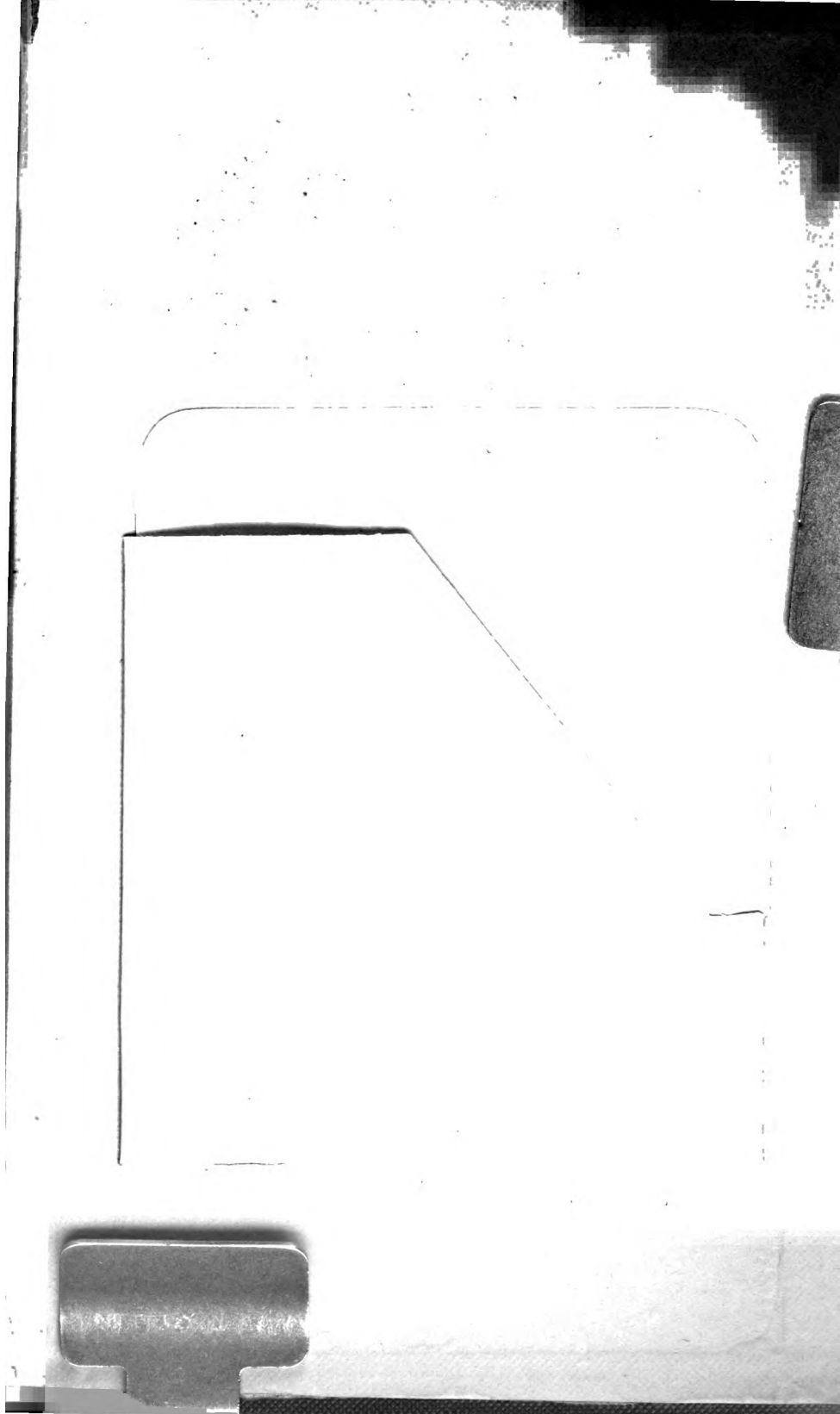
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